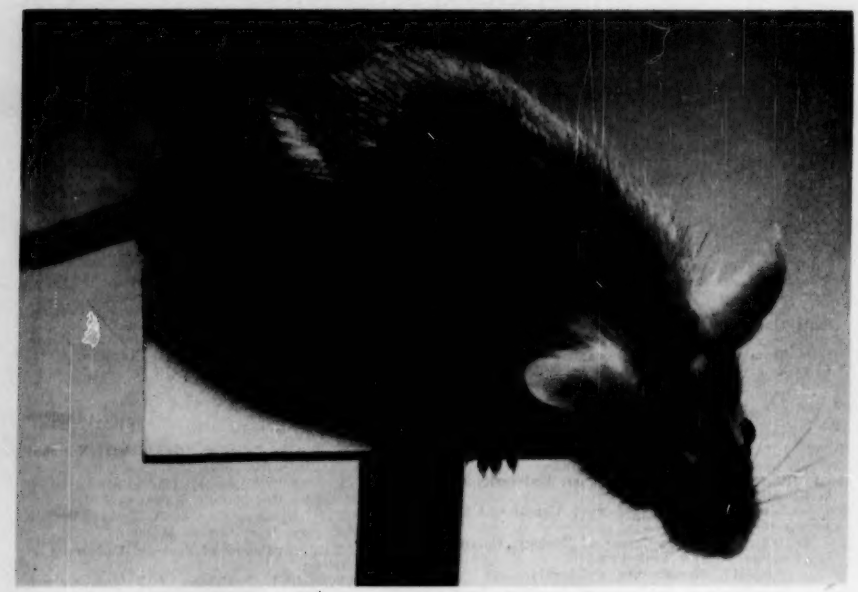
The Journal of HEREDITY



TUFTED—A NEW RECESSIVE MUTANT

(SEE PAGE 101)

Cytogenetics of Sugarcanes

Color Patterns in Leopard Frogs

Bicolor and Fuzzless Peach Sports

Russian Genetics Brought Up-to-Date

VOLUME 47 MARCH-APRIL, 1956 NUMBER 2

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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The JOURNAL OF HEREDITY is published bi-monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore 11, Md., and 1507 M St., N. W., Washington 5, D. C. Address all communications regarding editorial matters to 1507 M St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 29, 1918.

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$7.00 a Year, $1.50 a copy. Prices of back numbers on application.

The
Journal of Heredity

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N. I. VAVILOV

On April 2, 1956 Moscow news dispatches announced Dr. Vavilov's "scientific resuscitation." The account carries no indication that Dr. Vavilov is in position to enjoy a personal resuscitation. His death in Siberia in 1942 remains one of those irrevocable events which the rewriters of history cannot "correct."

Dr. N. W. Timoféeff-Ressovsky has recently returned from exile, with a mind apparently so warped by reindoctrination as to be beyond repair. For other casualties of the Lysenko era see page 55.

L'AFFAIRE LYSENKO: SPRING 1956

CONWAY ZIRKLE*

PRESS dispatches from Moscow dated April 10th, contained the news that Academician Trofim Denisovich Lysenko had, at his own request, been relieved of his duties as President of the All-Union Lenin Academy of Agricultural Science. The announcement was received by the geneticists of the world with much interest and some innocent merriment. A number of questions arose immediately: What was really happening to genetics in the Communist World? Had the application of "Michurin science" to Russian agriculture been as catastrophic as we had reason to believe and were the Communists about to rehabilitate Mendel and Mendelian genetics? Had the great prestige which American agriculture acquired so suddenly when the Russians visited our farmlands been responsible for Lysenko's removal? Or had the recent discovery by the Russians that the "cult of personality" was "inadmissible" been the cause of his downfall and was he demoted merely because he had been a protégé of Stalin's? These and other questions were calling for answers and the answers, if accurate, would tell us much about the biology which exists behind the Iron Curtain.

We have no lack of facts on which to base our conclusions but the facts do not fit neatly into any simple or logical framework. When judged by our own scientific standards or merely by the standards of simple intellectual honesty, they do not make sense. At present, we certainly cannot interpret them with any hope that our interpretation will be definitive. We have learned again and again that the words the Communists use to describe or justify their actions may be divorced from their usual meanings and that sometimes they are used simply as words—as words that have no operational meaning whatever. We should hardly expect that such denatured words would have any logical correlation with the acts they accompany. Thus, at any time, there may be an "Alice-in-Wonderland" quality in a Russian announcement, and this makes it difficult to discover just what they are up to. We know that, at times, they themselves do not know what they are doing.

Perhaps the best we can do so soon after the event of Lysenko's resignation is to list the pertinent happenings in a more or less chronological order and reserve our interpretations until after the tumult and the shouting have died.

There is no need to recount here the events which led to Lysenko's victory at the meeting of the Lenin Academy of Agriculture in August, 1948. These have been recorded many times and are readily available. Nor is there need to repeat the accounts of the first disasters which descended upon the more stupid of Lysenko's followers in the period immediately following his unprecedented triumph. These also are in our records. Nor need we even cite the evidence which shows that Lysenko was endorsed and backed by Stalin and Molotov.† Here we shall begin with the first direct attack on the triumphant Lysenko. It is significant that this attack occurred a year *before* Stalin died.

As soon as the western scientists refused to accept Lysenko's line in genetics and denounced him as an ignorant quack, the Communists began to suspect that they had made a serious error in placing all of their agricultural research in his charge. When their agriculture deteriorated and when Lysenko's great expectations were not realized, their suspicions were confirmed. Nevertheless,

*University of Pennsylvania, Philadelphia.

†Ivan D. London (Science 118:32. 1953) has translated Lysenko's letter to *Pravda* in which he admitted that Stalin had "corrected" his biology. Molotov had indorsed Lysenko in *Bol'shevik* No. 21, 1948.

for four years they sought to enhance his prestige and, during this time, they kept their propaganda machine at work protecting him from the "slanders" of the bourgeois geneticists and other reactionary and backward-looking biologists. From 1948 to 1952, *The U.S.S.R. Information Bulletin* (*VOKS*), contained numerous articles which recounted his virtues and achievements; but western geneticists remained unimpressed. Lysenko continued to demonstrate his naïveté and his inability to understand modern biology, particularly to understand the ratios of Mendelian genetics. This was very bad indeed because Lysenko was news, and western geneticists kept up with his pronouncements on genetical problems, and with the practices he advocated. Dobzhansky,* for example, reviewed one of his short works entitled "New in the Science of Biological Species," a hundred thousand copies of which had been distributed in the Communist World. This opus was stupid, even for Lysenko, and it seems to have been the first of his works which was attacked in Russia.

The Communist biologist who led the attack on Lysenko was Professor N. V. Turbin. Turbin had been one of the more pugnacious supporters of Lysenko in 1948. He is still a complete Michurinist and even if he supplants Lysenko, the Michurin doctrine will still be the official biology. In view of his present — and possible future — prominence, three short excerpts from his speech at the Lenin Academy of Agriculture are worthy of quotation.†

"Such works are characteristic of the most fanatical and orthodox adherents to Mendelism-Morganism who, with inflexible obstinacy and in the face of obvious facts, uphold even all those things in Mendelism-Morganism which the more farsighted representatives of this obsolescent theory are gradually discarding. . . .

"Michurin genetics attaches special importance to this category of facts, not because Academician Lysenko regards vegetative hybridization as the chief method of plant breeding, as some claim, but because vegetative hybridization is the chief and most striking proof of the unsoundness of the theory of the gene. . . .

"Many speakers here have already quoted facts obtained in experiments to transform winter plants into spring plants and vice versa. These facts show that it is possible by training plants under definite conditions of existence to obtain directed variations not only in their body characters, but also in their hereditary basis, if the nature of these characters has been sufficiently studied; that is to say, experiments have proved that acquired characters are hereditary."

Lysenko a "Mendel-Morganist"?

Turbin opened his offensive against Lysenko early in 1952 in the pages of the *Botanical Journal of Moscow*.‡ He accepted most of Lysenko's weird claims of having transformed one species into another, but he gagged at the statement that pine evolved into spruce and hornbeam into hazel. He did not accuse Lysenko of fakery at this time, but merely offered two alternative explanations: i.e., the supposed species changes could be accounted for (a) by invoking interspecific hybridization, or (b) multiparental pollination.

The adjacent paper in the journal, one by N. D. Ivanov, also attacked Lysenko (XXIII:1635). Ivanov criticized Lysenko's recent theories for their Mendel-Weismann-Morganist content and compared Lysenko's interspecific conversion to the mutation theory of formal genetics. Lysenko's errors, he asserted, consisted of the rejection of Darwin's concept of overpopulation, intraspecific competition and natural selection. He claimed, moreover, that Lysenko held views similar to Morgan's. We should note that Ivanov

*Th. Dobzhansky. Lysenko progresses backwards. *Journal of Heredity* 44:20-22. 1953.

†Proceedings of the Lenin Academy of Agriculture. Moscow, 1949. pp. 476-78.

‡*Plant Breeding Abstracts* has covered the entire debate in the technical journals. References to the abstracts will be given in volume and abstract number. Turbin's paper thus will be XXIII:1634.

actually rejects one of Lysenko's additions to Michurin "science," that of the non-existence of any intraspecific competition. Thus far, however, he seems to be alone in his daring. In fact, he went too far and had to retract (XXIV: 1571).

Lysenko's followers naturally rallied to his defense, for their own perquisites were at stake. The debate soon became truly fascinating, but here we can indicate only its broad outlines. None of the contestants were interested in anything so irrelevant as evidence derived from controlled experiments. They had much more effective weapons, such as good names like those of Darwin, Marx, Engels, Stalin and Michurin to apply to their friends; and bad names like Weismann, Mendel and Morgan to hurl at their enemies.

V. Dmitriev (XXIV:41) came to Lysenko's defense and rejected the criticisms because he said they were unscientific in that they conflicted with the principles laid down by the Soviet Darwinist and Marxist classics. O. B. Lepeshinskaya (XXIV:43) criticized Turbin and Ivanov for their disregard of Stalin's dicta on qualitative and quantitative evolution and for their denial of Lysenko's assertion that Darwinian evolutionary theory had some metaphysical elements. F. Dvoryankin (XXIV:48) criticized Turbin and Ivanov for their neo-Darwinist tendency. He regarded Lysenko's theory as rooted in Darwinism, but purged of Darwin's Malthusian errors.

Turbin also had allies. N. V. Pavlov (XXIV:42) agreed with him and Ivanov that Lysenko's new evolutionary hypothesis contained various elements characteristic of formal genetics. M. V. Kulik (XXIV:44) and E. V. Bobko (XXIV:45) rejected Lysenko's theory of conversion; and M. M. Iljin (XXIV: 788) stated that Lysenko's theories conflicted with the tenets of Marxism and Soviet Darwinism: that they even conflicted with Lysenko's earlier theories.

Some thirty abstracts were published in *Plant Breeding Abstracts* by April, 1954. The papers ran about two to one in favor of Lysenko. The biologists who owed their positions to Lysenko continued to rally to his support. After all, Lysenko's sun had been rising ever since 1928, and he had always been able to destroy any scientist he disliked. Before the year was out, the Soviet scientists clearly showed that they needed guidance if they were ever to reach a "correct" decision. Obviously, the political authorities would have to indicate what the "proper" course of action would be and which side of the dispute would be safe for the Soviet biologists to support. The procedure of enlightening the biologists followed the well-established pattern—the standard routine which had been tested many times. Only the minor details need ever vary.

First, a lengthy editorial appeared in *Kommunist*.* It charged that some scientists in the Lenin Academy of Agriculture, which Lysenko headed, were trying to monopolize science and that this was very bad indeed. It stated further that Lysenko's notions of evolution had never been subjected to a general discussion and that they needed airing. Finally, it criticized those who were trying to suppress the writings of N. V. Turbin. At this point, few biologists could miss the hint, and none could miss what followed. A Lysenko protégé was attacked publicly and demoted in such a way that it left no doubt as to who would win. It showed that Lysenko was no longer powerful enough to protect his sycophants. The protégé — incidentally a blatant incompetent who had been Chief of the Agricultural Planning Administration—was exposed in a letter to *Pravda*.† This letter gives a beautiful picture of Soviet practices:

"Several months ago, the Higher Certifying Commission asked me to evaluate a dissertation by Comrade V. S. Dmit-

**Science and Life.* Kommunist No. 5, March, 1954. pp. 3-13.

†S. Stankov. Concerning a faulty dissertation. *Pravda*, March 26, 1954. Translated in *Current Digest of the Soviet Press* VI:(12):13. 1954.

riev, doctoral candidate of the U.S.S.R. Academy of Science's Genetics Institute, 'On Primary Sources for the Origin of Certain Types of Weeds.' The basic idea of this dissertation is the assertion that cultivated plants themselves give rise to the weeds which plague them. It is claimed, for example, that rye engenders rye brome grass; oats, oat grass; sunflowers; sunflower broomrape, etc.

"After carefully familiarizing myself with this dissertation, I came to the conclusion that it could not be substantiated scientifically and was methodologically unsound. I therefore gave a negative judgment of this work and returned it to the Higher Certifying Commission.

"Comrade Dmitriev, the doctoral candidate, was also invited to a meeting of the commission's Biology Committee. He did not give satisfactory answers to questions from the committee members regarding methods of experiment. Moreover, the doctoral candidate displayed poor knowledge of the elementary laws of biology. And this is understandable, since V. S. Dmitriev, a candidate in economics, had not made a profound study of biology.

"After two discussions, the committee members formed an unfavorable impression of V. S. Dmitriev's dissertation. Trying to ease the situation, the committee passed a resolution to return the dissertation to the author for further work, although there was every reason to reject this research completely as patently erroneous.

"A meeting of the Higher Certifying Commission Praesidium was held February 13 under the chairmanship of Academician A. A. Blagonravov. After examining all the materials in the case of V. S. Dmitriev, the Praesidium recommended to a commission plenary session that it refuse the petition of the U.S.S.R. Academy of Science's Genetics Institute to approve V. S. Dmitriev for the academic degree of Doctor of Biology.

"Among other members present at the February 20 plenary session of the commission was Academician T. D. Lysenko, the academic advisor of doctoral candidate V. S. Dmitriev. Speaking at the plenary session three times, T. D. Lysenko undertook to defend V. S. Dmitriev's dissertation. In doing so, Academician Lysenko, with his customary sharpness, called all the reviewers who had spoken negatively of the dissertation, including myself, Weismannists. Academician Lysenko stated categorically that he would bear the responsibility for the high academic quality of V. S. Dmitriev's dissertation, without giving any reason for his conclusions.

"T. D. Lysenko's statements were supported by Academician A. I. Oparin, members of the V. I. Lenin all-Soviet Agricultural Academy V. P. Bushinsky and P. N. Yakovlev, and a number of others. The statements of these scientists were of general and declaratory nature, which is fully understandable, since none of them is a specialist in the field of botany.

"Despite the complete methodological and scientific unsoundness of V. S. Dmitriev's dissertation, the plenary session of the Higher Certifying Commission decided to confer the academic degree of Doctor of Biology on him. Sad as this may be, this fact itself testifies to an abnormal situation in our biological sciences.

"I am not a Party member, but have become accustomed to seeing justice embodied in our party. I deeply hope that justice will triumph in this instance also. After all, what I report in this letter can only be viewed as a mockery of Soviet science."

"Editor's Note.—Comrade Dmitriev's dissertation was again the subject of discussion at a meeting of the plenary session of the Higher Certifying Commission in connection with the receipt by the commission of additional data showing the scientific groundlessness and incorrect methods of research in this dissertation. After considering the additional data on V. S. Dmitriev's work, the Higher Certifying Commission resolved to refuse the petition of the U.S.S.R. Academy of Science's Genetic Institute to approve V. S. Dmitriev for the academic degree of Doctor of Biol-

ogy and to rescind the commission's decision of February 20, 1954."

The publication of this letter seemed to indicate that Lysenko's time was running out, and he appears now to be headed down the road he sent so many of his betters. He should not be liquidated violently, however, as this might cost the Soviets too much "face," and apparently they need all the "face" they can accumulate for the much more difficult task of telling the truth about Stalin.

Turbin returned to the attack and this time he accused Lysenko of fraud.* He stated that the evidence cited to prove that pine trees had evolved into spruce was clearly falsified, and the "evidence of the transformation of wheat into barley and oats, and rye into oats, lacked verification." In contradiction to his statement at the 1948 session of the Lenin Academy of Agriculture, he now rejects Lysenko's notion of 28-chromosome wheat being converted to 42-chromosome wheat, but interprets the transformation as due to an unstable hybridization of seed.

It is important that we note that Lysenko and his followers have at last been accused of fraud by the Communists themselves, and this leaves some British authors "holding the bag."† We do not know, of course, what he will do now, but it is worth remembering that, when Kammerer was exposed, he confessed and committed suicide. This need not establish a behavior pattern, however; Lysenko may simply vanish quietly in the orthodox Russian manner. But the Russians seem to be developing a newer technique for eliminating embarrassing individuals. They may simply remove Lysenko from all offices where he could damage genetics or agriculture and camouflage his scientific liquidation by giving him a gold medal or so. This technique might even replace their earlier and more savage customs. (But N. I. Vavilov will remain dead.)‡

Michurinism Sans Lysenko

But even if Lysenko loses all his honors and someone like Turbin comes into power Soviet biology need not recover. Turbin and the others who have attacked Lysenko are as much Michurinistic as Lysenko himself. They still use Weismannism, Mendelism and Morganism as though they were dirty words. Their biology is strictly Marxian with, of course, the Leninistic accretions. Thus far, their published work has shown no more scientific integrity than Lysenko's. No matter what doctrine ultimately takes the place of biology in the Communist World, it must always remain compatible with the basic Marxian postulates. Those who succeed Lysenko will have to conform to the basic pattern, will have to remain Marxian. Turbin may be the future great man. He is not as stupid as Lysenko, but he is committed just as much as Lysenko to "Michurin science."

On the other hand, the Communist authorities may consider Turbin to be expendable—useful for deflating Lysenko but not fit to succeed him. In fact, Pavel P. Lobanov, a Deputy Premier of Agriculture, now holds Lysenko's old position and Vladimir V. Matskevic, who led the Russian delegation to study the agriculture of the United States and Canada, has succeeded Lobanov. This latter appointment might indicate that the final decision to ditch Lysenko was reached after the Russians learned how superior

*_Journal of General Biology_ published by the Soviet Academy of Science. _The New York Times_ of July 6, 1954, describes the incident.

†Particularly those who have certified Lysenko's honesty, _e.g._, A. G. Morton (_Soviet Genetics_, London, 1951) and S. M. Manton (_The Soviet Union Today_, London, 1952).

‡The United Press on April 2, 1956 announced that N. I. Vavilov, who had been arrested in 1940 and who died in Siberia sometime in 1942, is being resuscitated scientifically, and that the U.S.S.R. Academy of Sciences is republishing his works in a new edition. When Lysenko replaced Vavilov in 1939, an attempt was made to make Vavilov into an "unperson." His name was dropped from the roll of both living and dead members of the U.S.S.R. Academy of Science and from all other records. He was too well known abroad, however, for such an attempt to succeed. The very attempt to make him an "unperson" had news value and backfired.

American agriculture is to their own. Indeed, Mikhail Spivak, the Minister of Agriculture, (*New York Times*, April 12, 1956) has said that many of the practices of United States agriculture are now being studied in Russia. During the year, Russia will import 500 tons of our hybrid corn of twenty different kinds for trial in the Ukraine. They have actually expressed their hope that, in the next two years, they will grow no corn there but hybrid.

It is even possible that the immediate cause of Lysenko's resignation is the success of Mendelian genetics in producing our hybrid corn. Kruschev himself has championed hybrid corn but Lysenko had made it impossible to produce such corn in Russia. (Spivak, however, said that Lysenko had not "violently" opposed hybrid corn but had merely wanted to limit it to research plots.)

There is no doubt that the Communist Authorities realize that American agriculture is based on sound scientific practices and that Lysenko's is not. But this does not mean that the Soviets will get rid of the Lysenko quackery completely. Those who make the final decisions in Russia are not scientists and their intentions are often better than their performances.

The Founder of Michurinism

When Lysenko succeeded in liquidating competent Russian geneticists and in outlawing Mendelian genetics, he replaced the geneticists with incompetent stooges and he replaced genetics with an aberration called "Michurin science." This complex of doctrines can be traced back to an uneducated nurseryman named I. V. Michurin who had been discovered by Lenin and made into an authority by Stalin. The degree of Michurin's biological sophistication can be judged from the following passage from his *Selected Works* (English translation, Moscow, 1949) from p. 207:

"In dealing with interspecific crosses definitely known to be difficult, I have often achieved success by adding a very small amount of pollen of the maternal parent to the pollen of the male parent."

This is what he actually wrote and it is one of the most simple-minded confessions in all botanical literature. It explains why these "successful" crosses all came within the seedling range of the mother plant. "Michurin science," however, is still respectable in Russia.

"Michurin science" does fit into the biological system endorsed by Marx and Engels in the 1870's and it will not be ejected from Russia easily. We have some evidence that indicates, that no matter how much the Russians may imitate our methods or how well their technicians succeed in increasing their agricultural production, the official biology will for some time remain that of Michurin. For one thing, there may be no one in Russia who has both the capacity and the official standing to attack Michurin science because there are almost no real geneticists left in Russia. For another thing, it would not be entirely without precedent for the Russians to say one thing and do another—to endorse Michurin science, but to follow the *ad hoc* American practices. No matter how much the Communists may claim "to unite theory and practice," the very existence of Communism often depends upon theory and practice being divorced.

Three recent instances show that the Russians, particularly the Russian biologists of the Lysenko period still do not realize that Michurin "science" is not science. The first one is described in an *Izvestia* news item in the issue of November 4, 1955, translated in the *Current Digest of the Soviet Press* (7:44:21), as follows:

"On November 3 (1955) the foreign scientists invited to the session of the All-Union Lenin Academy of Agricultural Sciences devoted to the One Hundredth Anniversary of the birth of I. V. Michurin, were received in the All-Union Lenin Academy of Sciences. Twenty-nine were scientists from eighteen countries who, as well as leaders of Soviet biological sciences, attended the reception.

"President of the Academy, Academician T. D. Lysenko, acquainted the

guests with the achievements of Michurinist biology and with the scientific problems on which our biologists are working. The scientists asked T. D. Lysenko a number of questions. The interview was held in a warm and friendly atmosphere.

"On November 3, Academician T. D. Lysenko spoke on topical questions of developing fruit growing in our country. . . .

"The U.S.S.R. Ministry of Agriculture has awarded the Michurin Gold Medal for 1955 to Academician T. D. Lysenko for his work in developing the teaching of Michurin."

The meaning of this gold medal and of the other things which have happened to Lysenko should be considered in the light of their timing. (1) Lysenko was first attacked early in 1952, a year before Stalin died. (2) Lysenko's protégé, Dmitriev, had his degree rescinded on February 20, 1954. (3) Turbin accused Lysenko of fraud on July 6, 1954 (*N. Y. Times*). (4) Lysenko was given a gold medal on November 3, 1955. (5) Lysenko's resignation was announced April 10, 1956. (6) Lysenko spoke at a dinner honoring Luther Burbank on April 11, 1956 and announced then that he is still Director of the Institute of Genetics, still a member of the Presidium of the Academy of Sciences, a member of the Academy of Arriculture Sciences, and a member of the Ukranian Academy of Sciences. (*N. Y. Herald-Tribune*, April 12).

Burbank as a Michurinist

The second instance has been called to the attention of western geneticists by Professor G. Ledyard Stebbins (see "New Look in Soviet Genetics" *Science* 123:721-722. 1956). Sometime in 1955, a book entitled "Hybrid Corn" (in Russian) was published by the Foreign Literature Publishers in Moscow. The work consists of translations of a number of articles by western geneticists. The geneticists, whose names are given by Stebbins, are Mangelsdorf, Shull, Hayes, Ritchie, Wellhausen, and Sprague. These had all contributed articles to "Heterosis" a book edited by John W. Gowen and published by the Iowa State College Press in 1952. Two aspects of this book "Hybrid Corn" are truly remarkable: (1) The name of the editor was suppressed; (2) The preface contained this statement, "Each of these articles contains much factual material; at the same time, since the authors of all the articles hold to the viewpoint of the chromosome theory of heredity, this material must consequently be used critically." The publishers obviously were hedging.

A review of this book by D. V. Lebedev was published in *Botanicheskii Zhurnal* in the issue of November-December 1955. The review begins by calling attention to a 1939 speech of N. I. Vavilov in which he told of the use of hybrid corn in the United States and how the utilization of hybrid vigor had greatly increased the yield. "The time that has elapsed since this address of N. I. Vavilov has demonstrated completely the basic correctness of the high value that he ascribed to the theoretical and practical knowledge of hybrid corn." (Vavilov, as we know, is no longer an "unperson"). The reviewer then states that the editor of the book is N. P. Dubinin. [Dubinin had been attacked viciously in *Pravda* September 2, 1947 and at the famous meeting of the Lenin Academy of Agriculture in July-August 1948. He lost his job as a geneticist but had been working as an ornithologist.] The reviewer held that the suppression of Dubinin's name as editor was inexcusable and that if "the chromosome theory of heredity" was invalid there was no reason for publishing the book. In fact, the reviewer made the caution of the publishers seem rather silly and showed that their fear of the Mendelian heresy was not approved by at least one person who was politically "correct" enough to write a review.

Our evidence at present indicates that the Russian politicians are trying to revive Mendelian genetics but that the Russian biologists are "dragging their feet." After all, the biologists are Michurinists and now hold positions of pres-

tige and comfort, positions that they would not like to lose. But they seem to be no longer able to maintain a complete monopoly in the biological sciences. According to a report from the editors of *"Drosophila* Information Service" M. S. Navaschin and N. P. Dubinin are now organizing genetics laboratories in Leningrad and Moscow respectively.

The third indication that the Russians are not aware that "Michurin science" is complete quackery is the fact that they translated the "Selected Works" of Luther Burbank into Russian. The translation was reviewed in *Izvestia* on January 10, 1956. (A translation of this review, which originally appeared in the *Current Digest of the Soviet Press,* February 22, 1956, is reprinted below.) The reviewer expressed the hope that the translation would bring the American and Russian scientists closer together. He may even have been sincere in expressing this hope for, undoubtedly, many Russians look upon Burbank as a great American geneticist and plant breeder. So do many Americans for that matter. The United States printed his face on a postage stamp. But Burbank was selected for this honor by politicians not by scientists.

Burbank was really the American Michurin. Most of his great "creations" were not his own at all but were merely new plants imported by the United States Department of Agriculture and given to him to market. It is possible that Burbank himself did not quite know what he had done or what his pollinations produced for he kept no adequate records. He neither understood nor accepted the contributions of Mendel, and he believed implicitly in the inheritance of acquired characters.

The Russians still have much to learn about western genetics. Their future education should be very interesting.

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RUSSIAN REACTION TO LUTHER BURBANK

Criticism and Review: LUTHER BURBANK. (By Prof. G. Eitingen, Doctor of Agricultural Sciences. *Izvestia,* Jan. 10, p. 3. 1900 words. Excerpts.)

In the days when our country and progressive people throughout the world were observing the centenary of the birth of Ivan Vladimirovich Michurin, the great Russian scientist who laid the foundation for a new stage in the development of the science of living nature, the Foreign Literature Publishing House in Moscow published a volume of the selected works of Luther Burbank (1849-1926)*. . .

The single-volume publication submitted for the reader's attention provides an adequate presentation of Burbank's methods and of his achievements in the field of pomology and the selection of trees, field and vegetable crops and ornamental plants. The book's preface, written by Academician N. V. Tsitsin, acquaints the reader with the landmarks in Burbank's work and contains a brief description of his views. . . .

One of Burbank's most interesting projects, to which he devoted 15 years of persistent work, was developing a gigantic cactus without spines. Thanks to its unique growth (it grew up to three meters in height and one blade weighed up to four kilograms), this cactus provided from 150 to 300 tons of fodder per acre for livestock. The improved cactus fruit tasted as good as the best oranges. Burbank made jam and candy from the cactus fruit.

In desert areas the cacti use their root systems to obtain moisture from deep layers of soil and store it during prolonged periods of drought. For this reason the cacti grown by Burbank are of great economic importance. Thanks to them, extensive dry, semiarid and arid areas can be transformed into habitable and productive ones.

Burbank conducted extensive experiments with corn, obtaining plants with ears that ripened ten days earlier than those of ordinary plants. . . .

As a convinced adherent of Darwinism, Burbank thought that the characteristics of plants arise under the influence of the environment and that new characteristics acquired under its influence can be transmitted. "I am personally convinced," he stated, "that every characteristic acquired by any organism be-

*L. Burbank, "Selected Works," translated from the English; general editor N. V. Tsitsin; Foreign Literature Publishing House, Moscow, 1955; 715 pp. Price 41 rubles.

cause of the influence of external conditions becomes a part of it and seeks to reproduce itself through inheritance." Following the teaching of Darwin, Burbank saw as one of the basic factors in evolution the variability of organisms, their ability to change to such an extent under the influence of the conditions of life that succeeding generations acquire new characteristics, which become hereditary. Thus inheritance, according to Burbank, is in the final analysis nothing but the sum total of all influences of the environment to which a plant is subjected during its historical development.

Burbank's views, which are fundamentally materialistic, are irreconcilably opposed to the theories of the Mendelians, who deny the possibility of the inheritance of acquired characteristics. Burbank's statements about Mendelian theory show this. For example, Burbank replied to one of his correspondents, who was attracted to the then "fashionable" Mendelianism: "I advise you to begin to study Mendel by reading Darwin; then have done with Mendel and read Darwin more thoroughly."

Burbank thought the main weakness of Mendel's theory and others like it was that they were at variance with the facts, and Burbank, like Michurin, valued facts above everything else.

Long years of hard work convinced Burbank that man possesses unlimited opportunities for transforming growing organisms in his interests. He thought that one of the most important reasons conditioning a substantial increase in the variability of organisms was the crossing of different parent forms, especially remote hybridization.

Burbank himself . . . achieved unique results in the field of hybridization, as well as in the use of selection of the best stock in developing new strains. But Burbank did not try to create, and did not create, a complete theory of the development of organisms.

Our great compatriot Ivan Vladimirovich Michurin created this theory by extending and developing Darwin's teaching. The theory is based on a study of the inviolate unity of the organism and the conditions of its life. Michurin performed the immortal service of thoroughly clarifying the role of the environment in the controlled raising of plants and animals from the early stages of their growth. . . .

The Soviet public and people in agricultural science and practice will greet the work of Luther Burbank with great interest. Publication of his work will also contribute to further strengthening the friendly ties between scientists of the U.S.S.R. and the U.S.A. and to their cooperation in the cause of utilizing the achievements of science for the good of the world and for the flourishing of peoples.

—*Current Digest of the Soviet Press*, February 22, 1956. Reprinted by permission.

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LEST WE FORGET

The "off-again-on-again" phase which Michurinist Marxist genetics seems to be going through at the moment has such comic opera aspects that we may be tempted to forget that Lysenko came to power under the tutelage of Stalin, whom even his former close associates now label a killer.

As Lysenko learned his genetics at Stalin's knee, so he seems to have learned some of Stalin's other techniques. Lysenko could not hope to equal his master's versatility as a purger, but in his modest way he did fairly well. For the record, a partial score is appended. This has been compiled from various sources, many of them confidential, so it carries no "literature cited." If evidence is forthcoming that any of these geneticists are alive and well, we will be happy to correct the record at a later date. We will also be interested in learning of any names which should be on this list, and which have been overlooked.—R.C.

Toll of the Missing

Leading Soviet Geneticists definitely known to be dead:

Agol
Executed as "traitor", 1936 or '37.

Koltzoff
Died, allegedly of natural causes, in early 1940's. Mrs. Koltzoff died a few days later.

Panshin
Shot as "traitor", 1945.

Karpechenko
"Died" in unexplained way during siege of Leningrad.

Kostov
Bulgaria; "died" during genetics controversy not long after returning from 1948 Stockholm Genetics Congress. It is believed but not sure that he was a brother of the executed, now reenstated, premier.

Vavilov
Died in Arctic Labor Camp at Magadan, probably 1942.

Those probably dead; not heard from or about since 1940 or earlier:

Kerkis
Lewitsky
Levit
Serebrovsky

Those not heard from or about since 1948 Condemnation of Genetics or earlier:

Alikhanian
Berg (Raisa)
Gershenson
Rapoport

Sent to labor camps prior to the Great Purges of 1936-'37:

Chetverikoff
Also his brother, a mathematician, later released under surveillance.
Ephroimson
Ferry
Lewitsky
Released at Vavilov's personal request to Stalin, but disappeared again later.

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STALIN AND MICHURINIST AGROBIOLOGY

By Academician T. D. Lysenko

The following excerpts originally appeared in VOKS Bulletin (No. 61, 1950) on the occasion of the Celebration of the 70th Birthday of J. V. Stalin.

The Leninist-Stalinist theory of developing agriculture on socialist lines and Stalin's personal leadership brought about a radical change in the life of the labouring peasantry of the Soviet Union in 1929. By uniting in collective farms, the Soviet peasantry took the only correct path as pointed out by Joseph Stalin, the path of Socialism.

* * *

It was the great Lenin who discovered Michurin and the great Stalin who launched Michurin's materialistic biological theories on the highroad of creative work. Progressive biology will always be grateful to Lenin and Stalin for supporting Michurin's teachings and providing all the conditions conducive to their development.

* * *

Not only has the great Stalin rescued the Michurin teaching from the attempts of reactionaries in science to destroy it; he has also helped to rear large forces of Michurinist scientists and practical workers. His guiding ideas have played and are playing a decisive role in the triumph of the materialistic Michurin teaching over reactionary, idealistic Weismannism-Morganism in the Soviet land.

* * *

The works of Joseph Stalin are an invaluable and inexhaustible fount for the development of theoretical Michurinist biology. His classic work *Dialectical and Historical Materialism* is an indispensable general theoretical aid to all agrobiologists, which helps them to gain a correct understanding of biological facts. Only when examined in the light of dialectical and historical materialism, the principles of which have been further developed by Stalin, does the Michurinist biological teaching gradually reveal its full depth and truth to us.

Stalin has furnished us with a powerful theoretical instrument which helps to disclose the laws of development of living nature. Thanks to this, Soviet biologists and agronomists can steadily advance the Michurin teaching in breadth and depth and thereby render increasingly effective assistance to the collective and state farms.

No one in the world has ever shown such great concern for science and for the verity and lucidity of scientific theory as have Lenin and Stalin. No one has ever done so much for science and for the well-being of the working people of the Soviet Union and the whole world as have Lenin and Stalin.

* * *

Only the materialist theory, raised to unprecedented heights in the works of Lenin and Stalin, has enabled biologists to develop Michurinist materialistic biology, which is free from all idealism, and thereby to perceive the development of living nature as a specific form of the movement of matter, to know living nature and its laws such as they really are.

* * *

A correct understanding of vitality is important both to theoretical biology and to practical plant growing and animal breeding. In the practice of seed-growing, proper intervarietal and intra-varietal cross-breeding yields seeds which produce more vital, hardier plants. Cross-breeding produces similar results in practical animal husbandry.

* * *

Neither in appearance nor in the number of chromosomes in its cellular nuclei does this hard wheat plant differ from ordinary hard

(*Continued on page* 104)

A SECOND DIPLOPOD MUTATION OF THE FOWL

WALTER LANDAUER*

IN 1947 Taylor and Gunns[4] reported on a new lethal mutation of the fowl which they called diplopodia. The mutation had occurred in a flock of White Leghorn chickens. The principal phenotypic traits of the lethal-type embryos are represented by a syndrome comprised of polydactylism of the hand and foot, reduction in length of the long bones and shortening of the upper beak. The expression of polydactylism was variable. In the great majority of cases (over 75 percent) the first toes of both feet were replaced by a group of three toes, of which the innermost was the longest. In some instances (about 10 percent) four toes were found unilaterally in place of the first toe; a similar number of variants had, either on one or both sides, only two toes in place of the hallux. In addition, a few exceptional cases had the first toe in its normal position and a group of supernumerary toes above it. Asymmetry in the number of polydactyl toes occurred in about one-sixth of all cases, and the majority of these had the greater number on the right foot. The tarsometatarsi contained a variable number of accessory elements. The tibiae were greatly shortened and bent, the femora were reduced in length to a lesser extent. The changes in the hands were similar in nature to those encountered in the feet. The long bones of the wings were also shortened. Most of the diplopod embryos showed a shortening of the maxilla. In rare instances, diplopod chicks hatched and one reached maturity, but in 98 percent of 423 cases the diplopod embryos failed to emerge from the eggs. The condition was clearly brought about by a single, autosomal recessive gene substitution, but there was some evidence for the existence of modifying genes.

In 1947 Dr. Lewis W. Taylor kindly supplied us with birds carrying the diplopod lethal, and our observations on this stock agree in practically every detail with the report of Taylor and Gunns, quoted above. In 1951 we found in Black Minorca fowl, purchased from a commercial source, a lethal mutation with a phenotype very similar to that of White Leghorn diplopodia. A comparison of the two phenotypes and a report on their genetic relations is the purpose of the present account.

Phenotypic Variation

Morphologically, many of the Black Minorca and White Leghorn lethal embryos are indistinguishable. This is equally true for gross external appearance and for the comparison of alizarin-stained and cleared specimens. The range of phenotypic variation is, however, broader for the Minorca diplopod embryos. This is illustrated for polydactylism of the foot by the data of Table I. The great majority (80.6 percent) of the Leghorn diplopod embryos had six toes on each foot, i.e. the position of the first toe was occupied by three toes; in 9.7 percent of the sample each foot had five toes, in 7.0 percent there were five toes on one foot and six on the other and in 2.8 percent the number of toes was six and seven, respectively, on the two feet. This distribution does not differ greatly from that recorded by Taylor and Gunns, but our material gives no indication of a significant deviation from equality, according to left or right body side, among the embryos with asymmetry of toe number. Whereas among the Leghorn embryos the frequency distribution is somewhat skewed toward the low number of toes (5-5), the opposite, and to a marked degree, is true for the Black Minorca diplopod embryos. Five toes on both feet were found only as a rare exception, the incidence of the bilateral six-toed condition is much lower (33.9 percent as against 80.6 percent), there are relatively many (22.9 percent) embryos

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Supported by a grant from the American Cancer Society, on recommendation by the Committee on Growth, National Research Council.

FEET OF DIPLOPOD EMBRYOS

Figure 1

A shows the foot of a White Leghorn diplopod embryo. *B-D* shows the defect in Black Minorca chickens.

with seven toes on each foot (Figure 1*C*)—not observed in the Leghorn material—and a few embryos even had eight toes on one or the other foot, i.e. five toes in the hallux position. Among the embryos with unequal numbers of toes on the two feet, the larger number was somewhat more often on the right foot, but the differences were not significant statistically.

There were other ways in which the Minorca diplopod phenotype differed—in some of the embryos—from the Leghorn diplopod material. In Leghorn diplopods with six toes on each foot, the medial-most toe is, as Taylor and Gunns have pointed out, generally quite large, whereas the other two accessory toes are relatively small (Figure 1*A*). This condition

was frequently found in Minorca diplopods as well (Figure 1*D*), but often the three toes were of almost equal length (Figure 1*B*). Syndactylism of the normal third and fourth toe was not a rare event among Minorca diplopods, especially in embryos with more extreme abnormality of the foot, (Figure 3); syndactylism did not occur in non-diplopod Minorcas. The polydactyl condition of the hands also exhibited in Black Minorca diplopodia a tendency toward more complex structural abnormality than that found in the Leghorn mutation, but these differences of expression are much less pronounced than those obtaining in the feet. Shortening of the upper beak frequently was more pronounced in Minorca than in Leghorn diplopod embryos. Finally, body size of the Minorca diplopod embryos tended to be smaller than that of the Leghorn diplopods, and death seemed to occur somewhat earlier. The 1954 data for mortality of the diplopod embryos in actual numbers were as follows:

	Mortality during incubation days	
	14-17	18-22
Leghorn diplopod	4	60
Minorca diplopod	20	79

Hatched chick and embryos living on the 22nd day were included in the 18-22 day group. The χ^2 is 4.923 with a P value of .03. The data of the 1955 season showed the same trend, but the differences were not significant. It must, in any event, be kept in mind that the ranges of phenotypic expression of the diplopod mutation of White Leghorns and Black Minorcas overlapped widely.

Crossing Results

We turn now to the question of genetic transmission. Tables II and III give the data for embryo mortality, hatchability and incidence of diplopodia (among survivors of the 13th day of incubation) in progenies of *inter se* matings of White Leghorn and of Black Minorca parents heterozygous for diplopodia. The Leghorn data for 1953 and 1954 show an incidence of diplopodia which agrees closely with the expectation for segregation of a single recessive gene. There was an appreciable

FROM HETEROZYGOUS PARENTS

Figure 2

A—Right foot of embryo one of whose parents was heterozygous for both, the other for one diplopod gene. The left foot had the normal four-toed structure. *B-D*—Feet of embryos both of whose parents were heterozygous for the Leghorn as well as the Minorca diplopod gene. *B* shows small excrescence (arrow) at the base of the first toe—the only indication of abnormality.

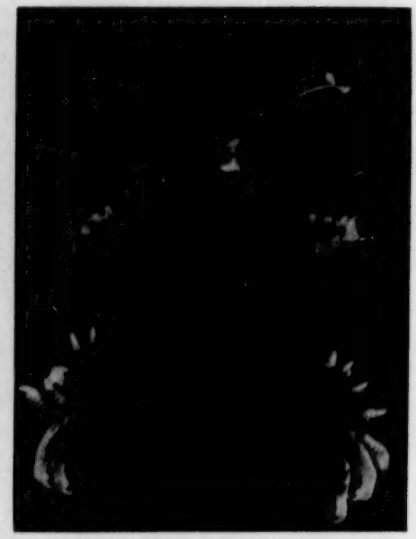
TABLE I. Distribution of number of toes in diplopod embryos from White Leghorn and Black Minorca stock and from *inter se* matings of birds heterozygous for both mutations

Number of toes: left — right	72 White Leghorn diplopod embryos %	118 Black Minorca diplopod embryos %	51 diplopod embryos from double-heterozygous parents %
4+ — 4	0	0	2.0
5 — 5	9.7	0.8	7.8
5 — 6	2.8	3.4	3.9
6 — 5	4.2	4.2	3.9
6 — 6	80.6	33.9	47.1
6 — 7	1.4	22.0	7.8
7 — 6	1.4	10.2	11.8
7 — 7	0	22.9	13.7
7 — 8	0	0.8	2.0
8 — 7	0	1.7	0

deficiency of diplopod embryos in the 1955 matings; at the same time early embryo mortality was exceptionally high, hinting at preferential mortality of diplopod embryos in a family which showed some detrimental effects of inbreeding in other respects. In the Minorca matings the incidence of diplopod embryos in each of the three years fell somewhat short of the 25 percent expected for the homozygotes of a segregating recessive gene, but the deviations are not significant for any of the years, nor for the cumulative data. It seems justified, therefore, to conclude that in both breeds we are dealing with a simple recessive mutation.

During 1953 we crossed five Black Minorca hens, heterozygous for diplopodia, to a White Leghorn cock who was also known to be heterozygous for the diplopod mutation. Among a progeny of 132 survivors of the 13th day of incubation none was diplopod. More extensive data were collected in 1954 and these are shown in Table IV. In these reciprocal matings between White Leghorn and Black Minorca fowl, known to be heterozygous for diplopodia, no diplopod embryos were found among 393 survivors of the 13th day of incubation. In only 3.2 percent of the 406 fertile eggs did the embryos die prior to the 13th day. It seems clear, therefore, that the diplopod variations of the White Leghorn and Black Minorca breeds represent discrete mutations.

It seemed of considerable interest to determine whether the genes that produce the two diplopod phenotypes are alleles. It was, therefore, planned to arrange matings of birds all of which were heterozygous for both diplopod mutations. In order to obtain such double heterozygotes, male and female offspring of the reciprocal matings shown in Table IV were tested consecutively with known heterozygotes of both types. In this manner we secured six females and one male which were heterozygous for both diplopod genes. When these birds were bred to each other, they produced the F_2 results shown in Table V. Fertility was, fortunately, nearly complete (see Table V).



22-DAY OLD DIPLOPOD EMBRYO

Figure 3

The mother of this diplopod embryo was heterozygous for both diplopod genes, the father was heterozygous for the Minorca diplopod mutation. The right foot has seven toes. The left foot has nine toes, of which four form a central complement among the toes in the hallux position. Down was removed from wing tips to show supernumerary fingers. The upper beak is short. Note syndactylism between normal third and fourth toe of both feet.

If the two genes for diplopodia were alleles and the double heterozygote is normal, as is known to be the case, we should expect equal

TABLE II. Progenies of *inter se* matings of White Leghorn fowl, heterozygous for diplopodia

Year	Fertile eggs	Mortality during incubation days 1-6 %	7-13 %	14-17 %	18-22 %	Hatched %	Survivors of 13th day Actual	Survivors of 13th day %	Diplopod among survivors 13th day %
1953	147	10.2	8.8	4.1	47.6	29.3	119	81.0	25.2
1954	286	4.2	9.8	1.7	30.8	53.5	246	86.0	26.0
1955	348	12.4	16.4	14.4	32.8	24.4	249	71.6	17.3

TABLE III. Progenies of *inter se* matings of Black Minorca fowl, heterozygous for diplopodia

Year	Fertile eggs	Mortality during incubation days 1-6 %	7-13 %	14-17 %	18-22 %	Hatched %	Survivors of 13th day Actual	Survivors of 13th day %	Diplopod among survivors 13th day %
1953	118	9.3	4.2	9.3	30.5	46.6	102	86.4	21.6
1954	467	3.6	3.4	4.1	22.7	66.2	434	92.9	20.0
1955	363	17.4	2.2	5.0	31.1	44.4	292	80.4	23.6

DIPLOPOD MUTANT

Figure 4

Feet of an embryo one of whose parents was heterozygous for both, the other for one of the diplopod mutant genes. *A*—In the right foot the hallux position is occupied by three toes. *B*—In the left foot three supernumerary toes are located above the normal hallux.

numbers of normal and of lethal-type embryos in the progenies of double-heterozygous parents. Actually, there were 120 normal and 51 diplopod embryos among 171 survivors of the 13th day. On the basis of a 1:1 expectation, $\chi^2 = 6.664$, P = .01. The hypothesis of allelism can, therefore, be excluded. Hence it is suggested that the diplopodia gene found in White Leghorn fowl be designated as *dp*-1 and that of Black Minorcas as *dp*-2.

If the recessive genes for the two diplopodia mutations were completely independent, the F_2 progeny should show a 9:7 ratio, viz., nine normals (including the double heterozygotes), three Leghorn diplopods, three Minorca diplopods, and one homozygous for both diplopod mutations. Should the double homozygotes be inviable or perish too early to be found among embryos that had died after the 13th day of incubation, a 9:6 ratio might be expected to appear in our data.

It seems difficult to reconcile our actual results with either one of these alternatives. For a 9:7 expectation, the data yield a chi-square of 6.424, with a probability of .01. A 9:6 ratio, it is true, is not ruled out by our segregation data ($\chi^2 = 3.896$, P = .05). If, however, the actual situation were represented by a 9:6 ratio, one should expect to find the double homozygotes among the embryos which died prior to the 13th day of incubation. Yet, if every one of the embryos that had died early is assumed to have been homozygous for both diplopod genes (and this is a very improbable hypothesis), the data still do not fit a 9:7 ratio ($\chi^2 = 5.011$, P = .03).

Discussion and Conclusions

It must, therefore, be concluded either that the normal segregation ratio is obscured by the fact that some diplopod homozygotes fail to express their phenotype and, as a consequence, are included in the normal group, or that independent assortment actually does not occur, i.e., that the two diplopod genes are on the same chromosome. This quandary cannot be finally resolved from information available at present. A few words may, however, be said with reference to the possibility of failure of the diplopod phenotype to be expressed.

The range of phenotypic variability makes it appear unlikely that normal overlaps occur either among the White Leghorn or the Black Minorca diplopods. In dominant polydactylism (*Po*+), in which occasional non-expression is a well-recognized fact, one finds graded steps blending into the normal phenotype. No such approach to the normal morphological structure has ever been reported in either one of the two diplopod mutations. The situation is different, however, in progenies of double-heterozygous parents. The last column of Table I shows the distribution of phenotypes (in number of toes) for these matings. Among the 51 F_2 diplopod embryos were two which

are of particular interest here. One showed a barely visible toe rudiment, a mere excrescence at the base of the normal first toe of the left foot (Figure 2*H*); the right foot was similar to that shown in Figure 2*A*, with one large supernumerary toe near the tibio-tarsometatarsal joint; the long bones, wings and beak were normal. The second embryo had five toes on each foot; on the left side the supernumerary toe was near the tibio-tarsometatarsal joint (Figure 2*D*); on the right foot the accessory toe, just above the normal first toe, was only loosely attached to the tarsometatarsus (Figure 2*C*) and might easily have dropped off; the long bones, wings and beak were normal. These are close approaches to the normal phenotype and the occurrence of entirely normal overlaps is, therefore, a distinct possibility in this group.

On account of the smallness of the sample, it is likely that the range of phenotypic expression of diplopodia among progenies of double-heterozygous parents is imperfectly represented by the data shown in Table I. This is indicated by the fact that additional, and very unusual, variants were found among the offspring of matings in which one parent was heterozygous for both, the other for one of the diplopod mutants. These included one diplopod embryo with four normal toes on the left and six toes on the right foot, and two embryos with nine toes (i.e. six toes in the hallux position) on the left and seven on the right foot (Figure 3). The occurrence of these variants suggests that the range of expression among F_2 diplopod embryos exceeds that of the White Leghorn and Black Minorca diplopods in both directions.

An occurrence of more extreme forms of diplopodia than that shown by either one of the two original mutants was to be expected in the F_2 progenies if there is a cumulative effect in the double homozygotes (*dp-1dp-1 dp-2dp-2*). The absence of a morphologically clearly-circumscribed class of extreme variants and the appearance of such exaggerated conditions in the progenies of *dp-1dp-1 dp-2* + × *dp-1dp-1* ++ or ++ *dp-2dp-2* birds and of + *dp-1 dp-2dp-2* × *dp-1dp-1* ++ or ++ *dp-2dp-2* fowl indicates that in zygotes homozygous for one of the two mutant genes the presence of the second diplopod gene in single dose is (or may be) responsible for a cumulative effect. This is interesting in view of the fact that the double heterozygotes are entirely normal.

Our observations demonstrate that the diplopodia mutation of Black Minorcas is a fairly close mimic of that found in White Leghorn fowl. It remains obscure how much of the higher degree of expressivity of the Minorca diplopod condition is produced by an actual difference in gene activity and how much may be accounted for by dissimilarities of residual heredity. We observed recently that the Minorca stock in which the new diplopodia mutation was found, also produces sporadic instances of shortening of the upper beak. This abnormality is independent of the diplopod condition and apparently is caused by multiple recessive genes. It was found, further, that treatment with teratogenic agents which are specific for shortening of the upper beak, results in a much higher incidence of this ab-

TABLE IV. Embryo mortality and hatchability in progenies of reciprocal matings between Black Minorca and White Leghorn fowl all heterozygous for diplopodia

Mating	Fertile eggs	Mortality during incubation days 0 %	7-13 %	14-17 %	18-22 %	Hatched %	Diplopod
Minorca ♀♀ × Leghorn ♂	261	2.7	0.8	0.4	6.9	89.3	0
Leghorn ♀♀ × Minorca ♂	145	2.1	0.7	0.7	3.4	93.1	0

TABLE V. Results of mating ♂ 6527 to six females, all seven birds being heterozygous for the Leghorn and the Minorca diplopod mutation

Mother	Fertile eggs*	Mortality during incubation days 1-6	7-13	14-17	18-22	Hatched	Diplopod
3059	8	1	0	0	1	6	0
3069	42	0	1	1	15	25	12
3115	20	1	1	1	6	11	7
3119	26	0	0	2	5	19	5
3122	46	1	0	2	24	19	16
3124	35	1	0	1	13	20	11
Total	177	4	2	7	64	100	51

* A total of 183 eggs were laid. Only six of these were infertile. All others are included in the tabulation.

normality in the Black Minorca than in White Leghorn stock[3]. It may well be that the same genetic liability accounts wholly or in part for the more extreme maxilla defect of Minorca as compared with Leghorn diplopod embryos. This reasoning may apply equally to other parts of the diplopodia syndrome, although genuine differences of gene activity between the Minorca and Leghorn mutant genes remain a distinct possibility.

There are two dominant and allelic mutations of the fowl, polydactyly and duplicate, whose phenotypes show preaxial polydactylism. It has been pointed out with reference to both (Landauer[1,2]) that it would be erroneous to interpret these gene effects as localized duplications of the hallux or as simple replacement of the first toe by a more complex complement of digits. In both instances we have found evidence for the conclusion that one is dealing with an incipient preaxial duplication of the whole foot. These duplications probably arise on the basis of quantitative variations in the tissues forming the foot or the whole extremity. We believe that these conclusions hold as well for the diplopod mutations. Many of our observations suggest that any part of the preaxial border of the foot may be affected by the diplopod genes. An additional illustration of this kind is shown in Figure 4*A* and *B*, representing an embryo which had the usual diplopod condition on the right foot (Figure 4*A*), whereas the left foot had a set of three supernumerary toes above the normal first toe. It is, therefore, not a question of morphogenetic alteration in formation of the hallux, but one of organization of the foot.

Summary

A new mutation has been found in Black Minorca fowl, the morphological traits of which closely resemble the diplopodia phenotype of White Leghorns previously described. Both mutations produce a syndrome of abnormalities the principal features of which are a peculiar form of polydactylism in both pairs of extremities, reduction in length of the long bones and shortening of the upper beak. In many instances the Minorca lethal-type embryos are indistinguishable from the typical representatives of Leghorn diplopodia, but in other specimens the Minorca mutants tend to be somewhat more extreme in their deviations from the normal morphological pattern.

The new diplopodia of Black Minorca fowl is inherited as a simple, autosomal recessive trait, as is the diplopodia of White Leghorns. In both instances nearly all diplopod embryos die during the last week of incubation; only as rare exceptions do such chicks survive to hatch. The two mutations are discrete and non-allelic. The symbol *dp*-1 is suggested for White Leghorn diplopodia and *dp*-2 for its Black Minorca mimic.

F_2 progenies from heterozygous Leghorn and Minorca fowl are deficient in the expected number of diplopod embryos. The cause for this deficiency must be sought either in the occurrence of normal overlaps among genetically diplopod embryos or in the existence of linkage between the two genes for diplopodia.

The double heterozygotes of Leghorn and Minorca diplopodia are entirely normal, but embryos which are homozygous for one and heterozygous for the other mutation or homozygous for both may show a phenotypic expression of diplopodia more extreme than that observed in either one of the two original mutant forms.

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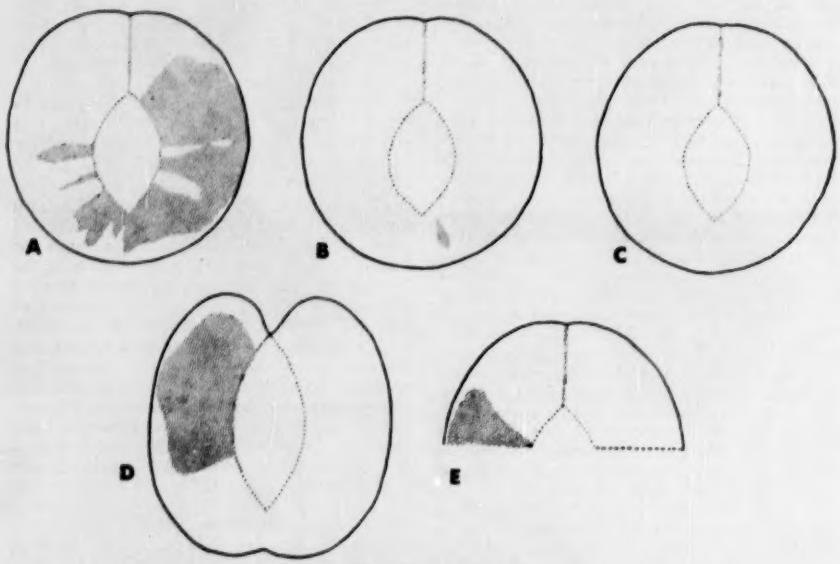
HISTOGENETIC FACTORS IN COLOR AND FUZZLESS PEACH SPORTS

HAIG DERMEN*



BICOLORED SPORTS

Figure 5

Diagrams of white and yellow chimeral fruits of a yellow-fruited Elberta variety sport. (See text for details.)

IN 1953 a study was made of two peach sports. One was characterized by a change in fruit color from yellow to white and the other by a change from normal fuzzy or pubescent fruit skin to fuzzless or smooth nectarine-like skin. These are separately described in some detail and their resemblance to some chimeral features in the fruits of cytochimeral peaches are discussed.

Description of Sports

Color sport. In 1949 Mr. and Mrs. Bruce Gallup, of Towson, Maryland, found partially white-fleshed fruits on a branch of an eight-year-old tree of yellow-fleshed variety Elberta. In September 1953, Mr. Gallup brought samples of these fruits to our laboratory for examination. Sketches of representative fruits showing the varying extent of the two flesh colors are shown in Figure 5.

The surfaces of the sport fruits lacked the characteristic yellow ground color of typical Elbertas and instead had the creamy pink surface color of the white-fleshed varieties. Internally, most of the sports were bicolored, with the areas of yellow and white varying in extent and pattern (Figure 5*A*), as in some types of leaf variegation.[3] In some fruits the flesh color was wholly white except for a yellow suture line (Figure 5*C*). The suture in the genus *Prunus* is the furrow formed along the fruit where the two edges of the carpel fuse. In other fruits

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MILLERS LATE PEACH
Figure 6

A shows a normal immature fruit; *B* a fruit with colored suture. Below these are shown the internal conditions of the two fruits. An arrow in *D* points toward a narrow dark line, corresponding to the fruit suture in *B* which was colored. There was no such distinguishing internal suture line in the normal fruit shown in *C*.

the yellowing extended in some areas from the skin to the pit (Figure 5*A*). In such cases there is presumably a narrow layer of white-colored tissue between the epidermis and the yellow area, but since the white layer is only a few cells thick, the line cannot be detected with the naked eye. The sketches in Figure 5*A* to *C* represent coloration range in transections of fruits. One fruit (Figure 5*D*) is shown longitudinally. On one side of this fruit there was a large yellow area separated from the skin at one point by a narrow strip of white flesh; the greater portion of this fruit was white. Figure 5*E* is a sketch of a transection through the middle of the half fruit shown in Figure 5*D*.

NECTARINE-LIKE FRUITS
Figure 7

This tree was propagated from nectarine-fruit bearing sport branch of the J. H. Hale peach variety. Professor Vierheller is pointing with one thumb and a pencil to two nectarine-like fruits. Most branches of this tree bore typical peaches; on some others fruits were partly fuzzless, and a few fruits were almost or entirely fuzzless like a nectarine.

It shows the depth of yellowing and the yellow suture line (stippled).

Colored suture in the peach. Normally there is no distinct difference between external and internal color at the suture line in the peach. However, a number of cases have been found in the peach, as well as in the cherry and plum, in which the sports have a distinctly colored suture both inside and outside. Figure 6 illustrates a colored suture peach sport. Shown in Figure 6*A* is a normal fruit of a yellow variety, Millers Late. Figure 6*B* shows the sport with the distinctive colored suture. Figure 6*C* shows a section view of the normal fruit and Figure 6*D* is that of the sport showing the colored suture faintly (indicated by an arrow). In contrast (Figure 6*C*), the section of the normal fruit flesh is of uniform color. The sport of Millers Late was discovered because its fruits ripened earlier than those of the normal variety. When photographed, the fruit in Figure 6*A* was still immature and lacked normal outer color.

The suture in the white sport of Elberta (later referred to as the Maryland sport) was colored. A similar feature in another peach sport involving a change of color from white to yellow is described by Yeager and Meader.[11] (This sport is later referred to as the New Hampshire sport.)

"Nectarine" (fuzzless) sport. In August 1953, the writer heard about a nectarine-like peach sport from Arthur H. Thompson of the University of Maryland. Dr. Thompson, with his associates, has given a general account of this sport[9] which was found in the orchard of R. S. Dillon, Jr., at Hancock, Maryland. A small branch on a five-year-old peach tree of Shippers Late Red variety—considered by some to be J. H. Hale—bore smooth-skinned, typically nectarine-like fruits. About harvest time, 1953, Dr. Thompson, A. F. Vierheller (also of the University of Maryland) and I, visited Mr. Dillon's orchard for a close examination of this sport.

Mr. Dillon had had propagations made from the original sport branch, hoping that this sport might become a commer-

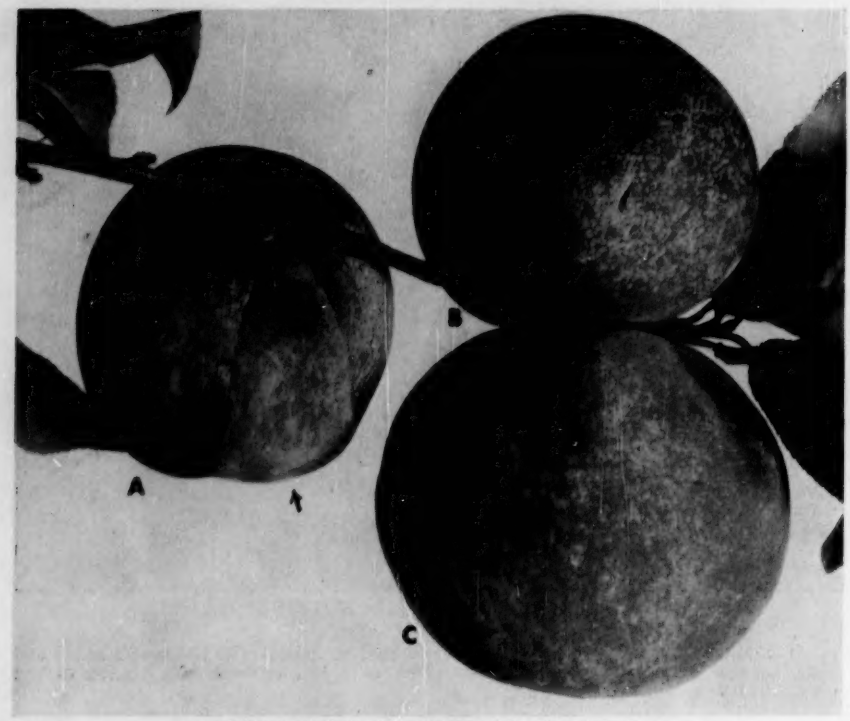
PEACH AND NECTARINE-LIKE FRUITS

Figure 8

The group of fruits in Figure 7 photographed at closer range. The two pencils at left and right, point to the two typical peaches; the other fruits were either entirely fuzzless or had very little hair in some restricted areas.

cially profitable variety of nectarine. In the process of pruning the orchard trees, the original sport branch had been cut off by mistake. At the time of our visit there were in the orchard, 11 trees, five years old, propagated from the sport branch. On five of the 11 trees about 30 percent of the fruits were partly smooth and partly fuzzy, and only rarely were entirely fuzzless fruits found. The remaining fruits were normal peaches and true to the variety.

One of the trees bearing fruit of both normal and nectarine type in Mr. Dillon's orchard is shown in Figure 7. Professor Vierheller is seen pointing to two nectarines. A clear view of the same group of fruits is shown in Figure 8, in which pencils can be seen pointing to the two peaches in the group; the other fruits were either entirely or almost entirely nectarines. Figure 9 shows three fruits attached to a twig from the same tree. Fruit *A* in this group was like a typically fuzzless nectarine except for a clearly defined sector along one side of the fruit suture (indicated by an arrow), the surface of which was raised and hairy. Fruit *B* was almost completely fuzzless, with short hairs over some restricted areas. Fruit *C* was mostly fuzzy, with limited areas where either the skin was smooth or the hairs were very short. Two detached fruits are shown in Figure 10. On fruit *A* a zigzag S shaped line separates the surface of the fruit into two portions; the left portion was fuzzy and higher than the rest, and the right portion was depressed and almost fuzzless. Fruit *B* appeared to be entirely fuzzless except for some areas covered with very short hairs. In general, the nectarine-type fruits were smaller, more highly colored than the normal, and the flesh texture was firmer: typical characteristics of true nectarines. If this sport could be made to remain truly nectarine, the fruits might be of good size, measur-



THREE TYPES OF FRUIT ON ONE TWIG
Figure 9

A—Nectarine-type fruit except for a band (indicated by an arrow) which is peach with fuzz; *B*—a nectarine-type fruit, smaller than a peach; *C*—a peach with some restricted areas either fuzzless or with short hairs. About natural size.

ing about 2½ inches or more in diameter. As pointed out later, it appears unlikely that this sport as constituted can be made into a stable form, suitable for commercial use.

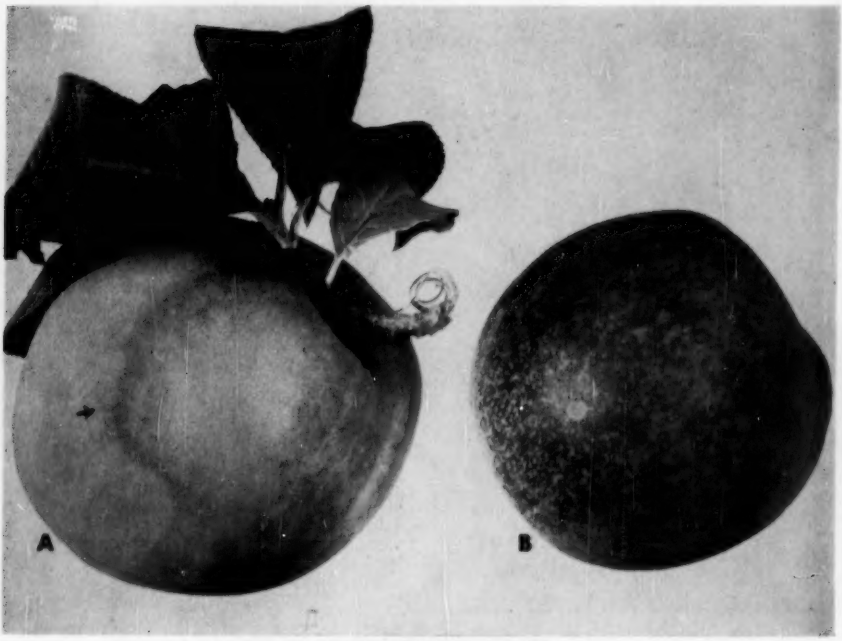
Histogenetic Interpretation of Sports

It was mentioned earlier that Yeager and Meader[11] reported finding a peach sport characterized by a change in color from white flesh to partial or total yellow. These fruits have a remarkable resemblance to those shown in Figure 5 except that the color change in the New Hampshire sport is the reverse of that in the Maryland sport described here. The change in both appears to involve the flesh under the skin, extending in some fruits to the pit in varying patterns and in others the change internally is total.

With change in color, there appeared within the fruits of both sports a straight line of coloration, which coincided with the suture in the peach. When homogenous white or yellow fruit is cut transversely, no distinctive coloration is seen along the suture line, either on the surface of the fruit or inside. In the New Hampshire sport the suture is white, bordered by yellow flesh. In the Maryland sport it is yellow, bordered by white flesh.

The fruits of the color sports show a close resemblance to those of certain types of cytochimeral plants, particularly with respect to the cytohistological development of the latter. To demonstrate the resemblance between the two classes of fruits certain details concerning the histological development in cytochimeral peaches are presented herein. There have been previous reports of certain cytohistological developments in fruits of the cranberry[3] and apple[5].

Colchicine-induced cytochimeras. Vegetative mutation may take place in any cell in any part of a plant. It may be perpetuated if it occurs in a growing point of a bud or a shoot tip. A mutation, whether due to



PEACH-NECTARINE SPORT

Figure 10

Fruit in *A* shows an S-shaped line (indicated by an arrow), which is peach on the left and nectarine on the right. The fruit in *B* is the nectarine sport. About natural size.

a change in a gene or to induced polyploidy, may occur in a single cell in any of the apical layers at a growing point or anywhere else in a plant. In a growing point the uppermost three cell layers, designated L-I, L-II, and L-III, are the most important, since all tissues of leaves, stems, and flowers are developed from them. A mutation occurring in a layer becomes part of the plant when a shoot is developed from the affected growing point and growth from such a shoot is maintained in some way, as through propagation.

Figure 11 shows a longitudinal section of a peach shoot tip in which L-II is tetraploid and L-I, L-III and other layers farther in are diploid. This figure shows cell layer formation in the shoot apex, illustrating a cytochimeral condition brought about by colchicine treatment[6]. Plants with this sort of chimeral makeup are designated as a 2-4-2 type, the numerals represent respectively L-I, L-II and L-III: two for diploid and four for tetraploid. Figure 11 shows—somewhat faintly—that the tetraploid L-II layer is characterized by larger cells and nuclei than other layers. Ink lines are drawn over and under L-II to set it off from the other layers. In colchicine-treated material of other plants either L-I or L-III was tetraploid and the other layers were diploid[6]. In the peach, as well as in the cranberry and apple, the origin of tissues was traced to the apical layers. This relation in the ovaries of two young cytochimeral peaches collected April 1, 1953, a few days after full bloom stage, is discussed here.

A transection of an ovary of one 4-2-2 peach is shown in Figure 12. In this ovary, the outer and inner epidermis and tissue a few cells in width at the suture line are tetraploid, while the inner tissue of the fruit is diploid. (Cytohistological development in ovules is excluded from the present study.) Two ink lines are drawn part way into the suture area and under the outer epidermis at both sides of the suture to set off tetraploid regions.

The ovary section shown in Figure 13 is from a plant representing a 2-2-4 type of chimera. The areas enclosed by black ink lines represent the tissue which is tetraploid; the areas outside of the lines are diploid. The white line marks the boundary of the tissue which differentiates into a pit. The pit boundary line was determined from a section of a young fruit collected at the so-called shuck stage (April 15, 1953). In the ovary at the

DIPLOID AND TETRAPLOID TISSUE
Figure 11

Longisection of peach shoot tip with a formula 2-4-2 (see text for details). Upper layer, L-I, is diploid; L-II is tetraploid; L-III and tissue below it is tetraploid.

stage shown in Figure 12 and 13 pit differentiation had not started. Figure 13 shows that tetraploidy in the ovary is not confined to a particularly defined region in this 2-2-4 plant. When the portion of the ovary toward the style end was studied, it was found that tetraploidy became restricted and was largely confined to the inner portion of the fruit. Tetraploid patches disappeared from certain areas and the amount of diploid area, on the whole, increased. On the other hand, when the parts of the fruit toward the stem-end were studied it was found that the tetraploid area was larger and the pattern of tetraploidy was less irregular.

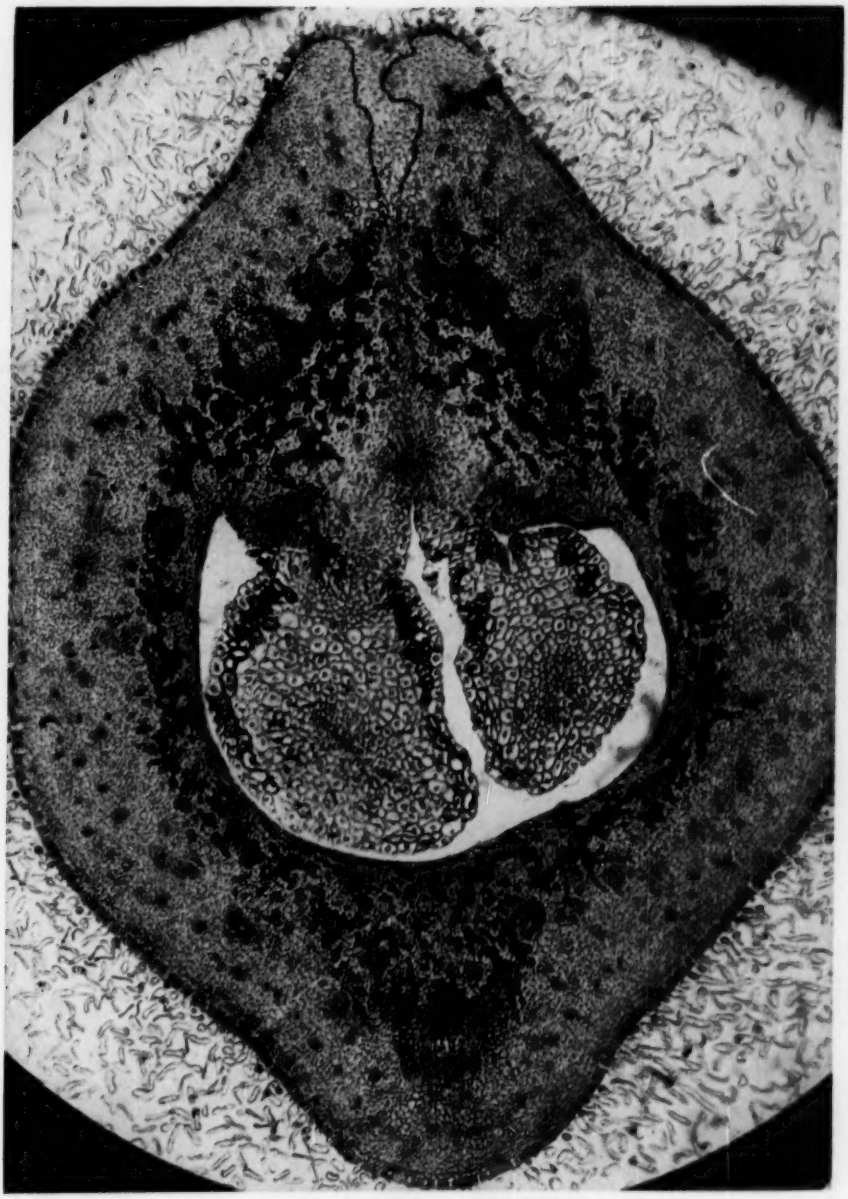
A small part of the fruit in Figure 13 is reproduced in Figure 14*A* showing more plainly that the cells and nuclei of tetraploid tissue inside the black lines in Figure 13 are larger than those outside. For comparison the same area is shown in Figure 14*B* without ink lines. To determine whether cells in an area are diploid or tetraploid, the size of the nuclei is the major factor to be considered. Cells are generally larger in tetraploid tissues, but they vary in size more than the nuclei, and are less dependable in determining the ploidy of the tissue. The broader band of tetraploid epidermal tissue in Figure 12 may be compared with the narrower band of diploid epidermal tissue in Figure 13. Other fruits of the 2-2-4 chimeral plant showed the internal tetraploid area in different patterns and amounts. Some fruits of this cytochimeral plant were entirely diploid.

Tissue more than two cells wide derived from L-I inside the fruit suture is shown in Figure 12. Sections of suture areas from three other ovaries are shown in Figure 15. Figure 15*A* illustrates the suture area in an ovary of normal diploid plants or in a 2-2-4 chimeral fruit as illustrated in Figure 13; the cells in the suture are all diploid. Figure 15*B* and *C* represent sutures in two ovaries in which only the epidermis is tetraploid as in Figure 12. The epidermal cells above the ink lines and the cells at the suture between the lines are tetrapoid. Larger cells and nuclei between the lines at the two sutures in Figure 15*B* and *C* are clearly apparent when compared with those outside the lines in the two sutures or in the suture area in Figure 15*A*. These size differences were determined under higher magnification, as in previous studies.[2,5,6]

Color sports and cytochimeras. In the color sport described herein and in the one reported by Yeager and Meader,[11] in most fruits the color of the tissue in the suture and in the tissue near the pit, was the same as that of the fruit of the normal mother plant. Studies of the cytochimeral fruits show that a limited amount of tissue in the suture is derived from L-I and that varying amounts of tissue around the pits are derived from L-III; the remaining tissues between the outer and the inner epidermis are derived from L-II.

When the diagram of the fruit in Figure 5*A* is compared with the illustration of an ovary section in Figure 13, a striking resemblance can be seen. This is seen in the patterns of the colored area in Figure 5*A* and of tetraploid areas outside the white line in Figure 13. As was indicated previously, in both types of fruits these areas varied and were extensive in some and greatly restricted in others. On the basis of the similarities just indicated, it may be assumed that in each color sport a mutation occurred in a cell in L-II alone and from it eventually a genetically distinct L-II layer evolved; initially it was limited to the apex of a single bud or shoot. The normal color in the suture indicates that L-I was normal and has given rise to normal colored tissue.

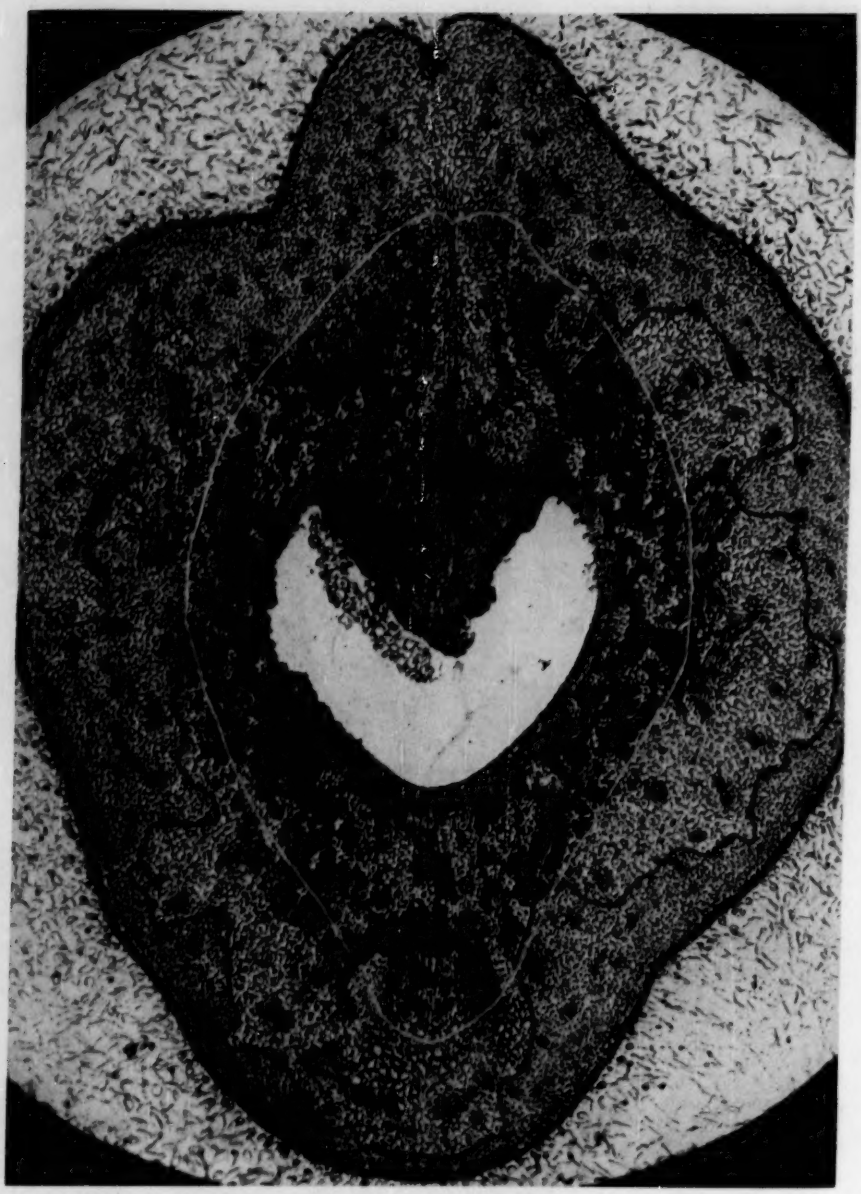
White peach flesh is genetically dominant over yellow, therefore the New Hampshire sport—white to yellow mutation—would represent a mutation of a dominant to a recessive gene, which is a common occurrence. In this case it may be assumed that the parent plant was heterozygous for color containing the dominant gene for white and the recessive gene for yellow. Hence, by mutation of the single dominant gene to recessive, L-II now carried



OVARY OF 4-2-2 CYTOCHIMERAL PEACH

Figure 12

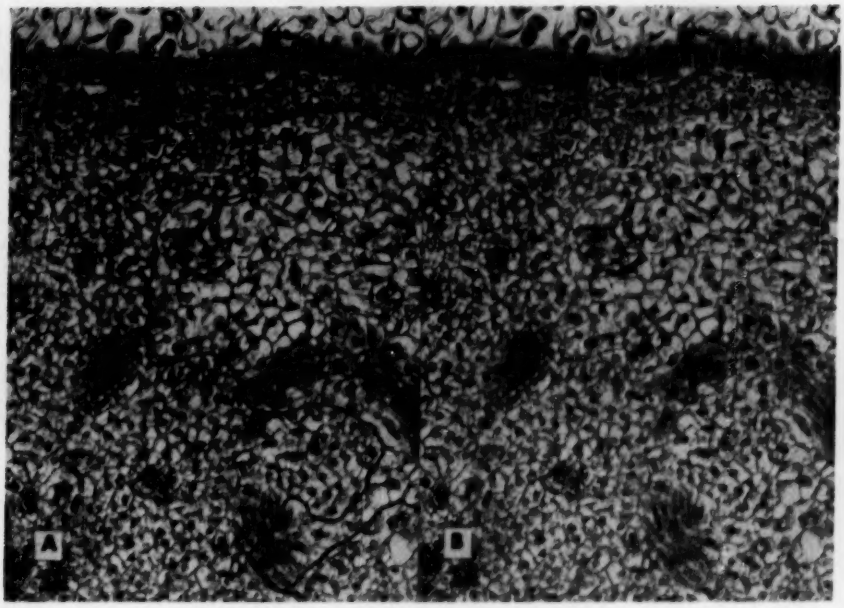
Epidermis outside and inside the ovary and the tissue at the suture (between two ink lines) is tetraploid. Remaining tissue in the ovary proper is diploid.



OVARY OF A 2-2-4 CYTOCHIMERAL PEACH

Figure 13

Tetraploid tissue is inside the black lines. Outside the black lines the tissue is diploid. The white line indicates the boundary of pit differentiation.



ENLARGED PORTION OF A 2-2-4 OVARY

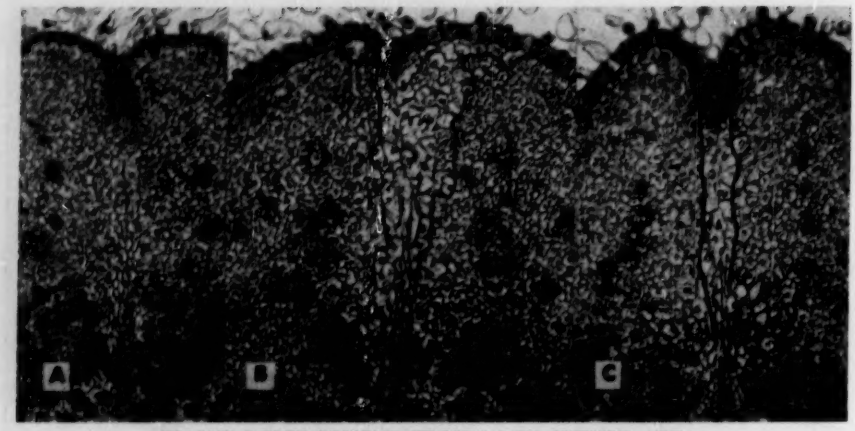
Figure 14

A—Enlarged portion of the ovary shown in Figure 13 (indicated by an arrow at left in Figure 13). Cells and nuclei inside the line, at right, are larger than those at the left. *B*—For contrast, the same area is shown without the ink line.

two recessive genes and the tissue developed from it appeared yellow. This particular line of events is indicated by the origin of the mother plant of this particular sport.[11] As reported by Yeager and Meader,[11] the white-fruited mother plant had originated from a cross between a yellow-fruited and a white-fruited plant. Therefore, in L-II of the bi-colored (yellow-white) sport there would be two recessive genes, and that would make the color of tissue derived from it yellow. On the other hand, it appears that a reverse mutation had occurred in the Maryland color sport. The Elberta variety from which this was derived has yellow fruit. In this case white represents a mutation from yellow, a rare case of a gene changing from recessive to dominant. The Maryland sport would represent a heterozygous condition for white and yellow. White being dominant, the tissue derived from L-II appeared white. In selfing, the New Hampshire type should produce only yellow-fruited progeny; the Maryland type should produce both white and yellow-fruited seedlings. These are to be expected since sex tissues are known to be derived from L-II.

The color sports appear to differ from the cytochimeras only by the fact that change of color is involved, making the fruits *bicolor*, whereas in the cytochimeral plants the change is due to polyploidy and fruits have become *biploid*. Developmentally the pattern formation in both has the same histogenetic basis. The difference in the pattern of tissues developed from L-II and L-III in the cytochimeral fruits was due to a variable rate of mitotic activity in different portions of tissues derived from the two cell layers. When there is considerably more mitotic activity in L-II and cells divide more periclinally than anticlinally, a larger extent of tissue in the fruit is derived from L-II and comparatively less from L-III and vice versa. By periclinal cell division in L-II all internal tissue in a fruit may be derived from that layer alone. When the internal tissue of a vegetative bud is entirely of L-II origin, then fruits developed on branches originating from such a bud, would bear only fruits of a genetic or cytological nature associated with L-II. This would explain why some fruits in the Maryland sport are internally all white-fleshed and in the New Hampshire sport internally all yellow, with the exception of the suture area.

Suture color and epidermal cytochimera. As was pointed out previously the epidermal sys-



TISSUE DEVELOPMENT FROM L-I IN SUTURE AREA

Figure 15

A—Suture area in an ovary of a normal diploid peach plant. *B*—Tetraploid tissue (between two ink lines) in the suture area several cells wide in an ovary of a 4-2-2 plant. *C*—Tetraploid tissue only two cells wide (between two ink lines) in the suture area in another ovary of the 4-2-2 plant. The tetraploid areas are bordered by diploid tissue.

tem, a single cell layered tissue, in the peach (also in the cranberry and apple) is derived from L-I. At an early stage of development the ovary is somewhat like a U-shaped body in transection. Later it becomes a closed system. Where the two edges of the primordial U-shaped body meet and a suture is formed, a minimum of two layers of cells in the suture are derived from L-I. As the ovary grows the cells at the suture divide generally at all angles as do other internal cells in the ovary. Consequently in transverse sections the tissue in the suture derived from L-I may be several cells wide (Figure 15*B*) and only rarely two cells wide (Figure 15*C*). In the color sports, the fruit color is determined by the color of the tissue under the epidermis. Only at the suture, both on the surface of the fruit and where deeper tissue from L-I developed internally did the color of the mother variety show. It seems, where tissue such as the epidermis, is only one layer thick, although its genetical nature may be different from that of the tissue underneath, it is too thin to ordinarily express color by itself and its color is therefore affected by the color of tissue underneath. Only when tissue developed from L-I is several cells thick does it show color when the color of tissue underneath it or adjacent to it is different. Yeager and Meader[11] made several diagrams of their sport, which in transection show distinctive white suture color in all fruits except one. This particular fruit actually lacked white suture color (personal communication from Meader). Apparent lack of color at the suture in this fruit may be explained on the assumption that perhaps tissue in that area was not thick enough to show color to the naked eye. Under a magnifying lens, perhaps, a white suture line might have been seen.

Fuzzless sport and cytochimeras. There have been numerous reports in the literature of somatic mutations from peach to nectarine.[7] It has been shown that the nectarine (fuzzless character) is recessive to the peach (fuzzy character) and that when a nectarine is crossed with a true breeding peach the first-generation seedlings are all peaches. According to Crane and Lawrence[1] in such a cross "there is no evidence that the smooth fruit character has been passed on to the progeny." Oberly and Nicholson[8] report a discovery of a nectarine sport on a peach variety and suggest that the parent peach variety may be heterozygous for the peach and nectarine characters.

The characteristics of the few completely fuzzless fruits of the sport described earlier appear to be similar to those of generally known nectarines. It has not been possible to determine whether the tree—or the limb—on which this sport occurred was homozygous for peach or whether it was heterozygous and carried one gene for the nectarine character along with one gene for the peach character and, by mutation, resulted in two recessive genes occurring in the sport shoot. The peach trees grown in Dillon's orchard are supposed to be of Shippers Late Red variety. However, according to Leon Havis, of this station, these trees are indistinguishable from trees of the J. H. Hale variety. Unpublished data obtained by Dr. Havis indicate that the J. H. Hale is homozygous for the peach char-

acter. Assuming that the parent tree is of the J. H. Hale variety, then it would seem that this fuzzless sport carries one dominant gene for the peach character and one recessive gene for the nectarine character. Theoretically, under these conditions the nectarine character should not be expressed. On the other hand, in the present case, at least, the mutation having occurred somatically, it may be that the nectarine gene behaves as a dominant, whereas in a normal breeding process it behaves as a recessive. It is clear that there is a need for genetic tests* for the solution of this apparent enigmatic problem which has puzzled plant breeders for generations.[7] It should be stressed that those who may attempt such a study should be aware of certain histogenetic features of the shoot apex in peach, as in many other plants, and their bearing on the mechanism of somatic mutation.[2, 4]

Since the discovery of the nectarine sport, it has not been possible to develop a stable form of it nor does it appear that this particular sport can be made stable. It seems probable that the sport occurred as a genic mutation and was carried in L-III in the branch originally discovered. This is indicated by the fact that after propagation, no single tree, either large or small limbed, has been found to bear entirely fuzzless peaches or nectarines. On the contrary, on trees that bore nectarine-type fruits, there were located many large and small branches which bore only typical peaches. Two points may be considered. (a) If the mutation had occurred in L-I, from which the epidermis is derived, all the fruits on trees propagated from the sport material would have been fuzzless, since all propagated trees would have the same genetically constituted epidermis. However, six of 11 trees propagated from the sport twig have borne only peaches. On the remaining five trees many branches bore only peaches. On others, many fruits were peaches and many were part peach, part nectarine; there were only a few fruits which could be classed as true nectarines, characterized by certain definite qualities: lack of pubescence, smaller size, firmer texture and typical nectarine flavor. (b) If this mutation had been carried in L-II and somehow affected hair development in the epidermis, then again all fruits should have been nectarines because the same L-I and L-II would have coexisted indefinitely in the propagated trees as long as cell division in L-I took place anticlinally, which is usually the case. Only rarely does periclinal cell division occur in L-I, as when a daughter layer of L-I replaces L-II. Because of such a process of replacement, some fruits would have developed as peaches in part or *in toto* instead of as nectarines, but the great bulk of the fruits would have been nectarines. The facts were against these suppositions.

On the premise that this sport is of a genic nature, it could not have been present in either L-I or L-II, it then appears most likely that the mutation occurred in L-III. As stated, the nectarine character is asscoiated with a recessive gene. If the mutation is the same or similar to that associated with the commonly known nectarine fruit, then such a recessive could not have been expressed somatically, since a dominant gene for hairiness would still be present. As has been pointed out, sex tissue has been shown to be derived from L-II. Since this mutation is presumably present in L-III, its genetic nature may remain unsolved. It seems probable that the effect of the mutation in L-III is exerted on hair development by some matter, possibly hormone-like, carried by diffusion from the tissue developed from L-III, into the epidermal tissue. Accuracy of the above explanation may be tested with the aid of two other kinds of nectarine sports, if and when they become available: one carrying the mutation in L-I and the other carrying it in L-II.

The fruits of this sport have shown certain features which resemble the development of the 2-2-4 chimeral fruits. In this cytochimera the great variability of tissue developed from L-III was demonstrated. In some parts of the ovary (Figure 13) tetraploid tissue developed from L-III was very close to the outer epidermis and in other parts either little tetraploid tissue was developed from L-III or the same kind of tissue was found deeper in the fruit. In some areas extending from the outer to the inner epidermis tetraploid tissue was entirely lacking. In some cases, however, L-III was replaced completely and thus internal tissue in the fruits was almost entirely of L-II origin and only occasionally of L-I origin.

Examination of fruits on trees propagated from the fuzzless sport revealed three types: (a) Most of the fruits were normal, and many branches bore only normal peaches; (b) in some fruits the extent of fuzzy and fuzzless areas varied; and (c) only a few fruits that looked typically nectarine were found. The following suggested explanation is based on these observations. (a) Completely fuzzy fruits represent cases in which the sport tissue is entirely absent, and in such fruits internal tissue would have developed from L-II and rarely from L-I. (b) In chimeral fruits, fuzzless areas appear where the tissue derived from L-III is close to the skin, and fuzzy areas appear where the tissue from L-III is either lacking or is farther away from the skin. Tissue from such areas at considerable depth or entirely lacking would be derived from L-II. (c) Completely fuzzless fruits

*The author would appreciate information on somatic sport of the nectarine on peach that has remained constant nectarine and has not reverted back to the peach. Such a nectarine mutation may have occurred in L-II and would be valuable for genetic analysis whereas the nectarine sport reported herein is not, since the mutation is presumably present in L-III.

would have almost the entire internal tissue derived from L-III, with only a narrow strip of tissue under the epidermis developed from L-II and with tissue in the suture being derived from L-I. A well defined sector on fruit *A* in Figure 9, situated right by the suture, may represent a case in which a band of underlying tissue much wider than usual (Figure 15*B*) was derived from L-I, which being genetically normal resulted in the development of a normal fuzzy condition.

Summary

Two peach sports, one showing change of flesh color from yellow to white and the other showing change of normal fuzzy skin to smooth nectarine skin, are described. The color mutation seems to be present in L-II in the shoot apex and the nectarine mutation in L-III. The majority of the fruits of this sport appear to be a reversion to normal fuzzy. Others are part nectarine and part peach. One unique feature of this fuzzless sport is that the nectarine character is ordinarily known by the breeding evidence to be controlled by a recessive gene. Here, however, it has appeared somatically as if the nectarine character were controlled by a dominant gene. Certain developmental features in the ovary of a 2-2-4 cytochimeral peach are described and histogenic relations between such a chimeral ovary and the sports are discussed.

Addendum

It has been suggested[4] that supposed graft-chimeras in some woody plants may be periclinal chimeras occurring naturally through some somatic mutation. Now and then mention is made in the literature of "graft-hybrids." It may be that in these cases the term "graft-hybrid" is used erroneously to mean "graft-chimera." If graft-hybrids are produced or discovered, (a) it would be assumed that union has actually taken place between two somatic cells, one from the scion and the other from the rootstock; (b) that the nuclei of the two cells have fused where scion and rootstock have made a union; (c) and that from the united cell has developed an endogenic bud which has grown into a branch. The course of these events would imply that the somatic hybrid thus originated contains the sum of the somatic chromosomes of the parent stocks. The graft-hybrid, then, would be a true amphiploid quite similar to tetraploid hybrids obtained from doubling the chromosome number of a diploid hybrid.

It is suggested that these supposed graft-chimeras and graft-hybrids be studied in the same manner that the peach-nectarine, bicolored chimeral sport and cytochimeral plants were studied and that the genetic nature of such plants also be analyzed. It may turn out that "graft-chimeras" and "graft-hybrids," particularly those of woody plants, are only periclinally constituted chimeral sports that have occurred through either genic or polyploid mutations. "Graft-chimeras"—not graft-hybrids—between species of *Lycopersicon* and *Solanum* were first produced experimentally by Winkler[10] and later by many others. There has never been a case reported in the literature of a graft-chimera in a woody plant produced experimentally. The term "graft-chimera" has a valid meaning when applied to the two genera of the herbaceous plants mentioned, but it should not be used with reference to woody plants, since so far as the author knows, there has never been convincing proof of its existence in woody plants. As to the term "graft-hybrid," I think it is fictional and has no place in scientific literature.

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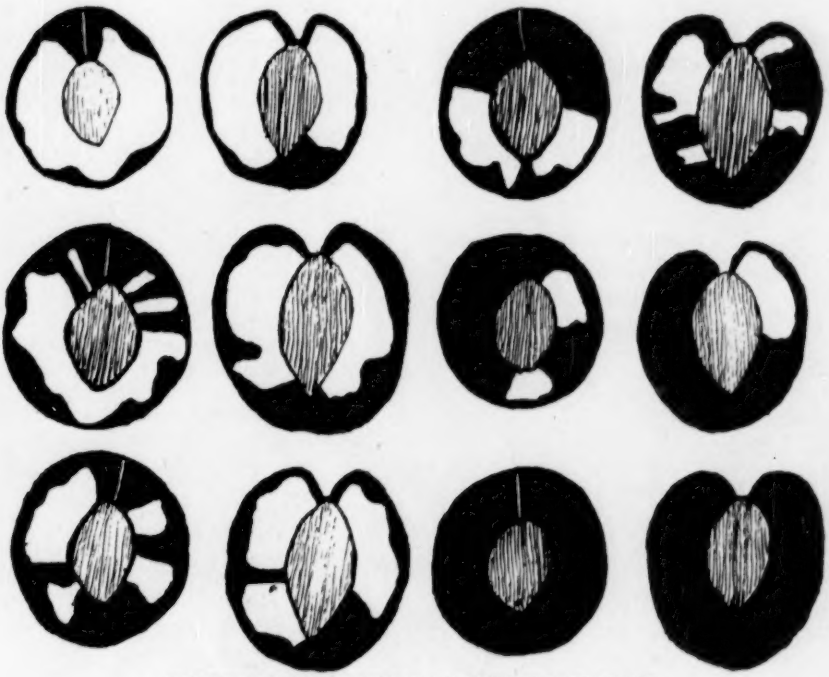
A FLESH-COLOR CHIMERA IN THE PEACH

A. F. YEAGER AND E. M. MEADER*



WHITE FLESHED AREAS IN YELLOW PEACHES

Figure 16

Six pairs of chimeral peaches showing a section cut across the middle at the left of each pair with the suture at the top. At the right in each pair is a longitudinal section of the same peach with the suture line at the back. The black area represents the yellow parts of the flesh.

IN 1949, three yellow peaches were collected from among many white-fleshed fruits which had fallen beneath a seedling tree. The tree had resulted from a cross between Eclipse, a yellow variety, and a seedling peach from the North Caucasus Region. The Caucasian peach itself bears small white-fleshed fruits. At the time, the small branch that had produced the yellow fruits could not be identified. Close watch was kept on the tree in 1950 and the small branch which again produced yellow peaches was identified and labeled.

Upon being cut, the three yellow peaches were found to have yellow flesh on the outside and white flesh near the pit. The distribution of the two colors was irregular being somewhat like the distribution of two flesh types in the Sweet-and-Sour Greening apple chimera described by Gardner[2]. There were only a few peaches on the branch in 1950 and all of them exhibited this characteristic color pattern within the flesh. As a re-

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sult of careful pruning to favor the sport, in 1951 the branch produced 83 peaches—all yellow in outward appearance. When cut, 23 fruits showed part white and part yellow flesh. Sixty fruits had yellow flesh except for a white streak extending from the skin to the pit at the suture line. No fruits having completely yellow flesh were noted. A count was not made in 1952 of the proportion of those with mixed flesh color and those with yellow flesh accompanied by a white suture. In 1953, of 100 peaches examined from the sporting branch, 11 showed a combination of yellow and white flesh; the distribution of each color being in the same irregular pattern. The rest were yellow except for the white suture. The fruits having this combination of yellow and white flesh were scattered throughout the bearing area and were not found to be located in any special part of the branch. It can be noted, however, that the percentage of the peaches showing some white flesh around the pit was less than that observed in 1951.

A single three-year-old tree which had been propagated by budding from the original sport produced more than 50 peaches in 1953. All showed solid yellow flesh extending to the pit, except that most of them had a clearly visible white line of flesh from the skin to the pit underlying the suture. This white suture resembles the condition found in the Rainbow Stripe cherry, a white variety which resulted as a sport of Lambert, a reddish-black-fleshed variety. The cherries of Rainbow Stripe[1] are white with the exception of the suture line which is blood-red. This narrow colored area of the flesh extends inwardly to the pit.

Since peach varieties are maintained as clones, the type of fruits that might be borne by trees resulting from individual buds selected for propagation from the yellow-white flesh chimeral sport has practical importance beyond an academic interest. How faithfully any one of the several flesh-color patterns listed might be perpetuated through propagation could only be determined by many years of careful and detailed study. Since small fruit size does not encourage commercial propagation, this may never be done. Nevertheless, the plant material is available at this station for distribution to anyone having an interest in further study of this chimeral flesh color in the peach.

Figure 16 shows the irregular distribution of the white-fleshed area in the peaches collected in 1951 and 1953.

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MUTANT COLOR PATTERNS IN LEOPARD FROGS

A Possible Multiple Allelic Series

E. PETER VOLPE*

GENETIC studies on amphibians have lagged far behind the embryological researches on this group. The analysis of hereditary factors in the common leopard frog, for instance, has received little attention from geneticists, and yet, the developmental mechanisms of this form have been analyzed extensively by embryologists. The principal reasons for this paradox lie in the low reproductive activity of the frog (i.e., breeds only once a year). in the difficulties encountered in rearing a large population of tadpoles through metamorphosis, and in the long period required by the offspring to reach sexual maturity.

In spite of the limitations of amphibian material, certain conspicuous variations, in a sense too striking to have remained unnoticed, have been subjected to genetical analysis. The white strain of the Mexican axolotl (Haecker[3]), the non-spotted condition in the leopard frog (Moore[6,7]), the striped color pattern in Ricord's frog (Goin[2]), polydactyly in the European toad (Rostand[9]), the fluid imbalance in the axolotl (Humphrey[4]), the ventral coloration in the Takata black type frog (Moriya[8]), and the mottled color pattern in the leopard frog (Volpe[12]) each represent the expression of a single mutant gene. The present level of genetical studies in amphibians is reminiscent of the early investigations in *Drosophila melanogaster* where easily discernible qualitative differences were examined and in most cases were found to be based on simple Mendelian inheritance. However, just as more complicating types of inheritance were unraveled in the fruit fly, it seems almost certain that a more extensive analysis of variant traits in amphibians would reveal complexities in genic action. The present report deals with the genetical interrelations of three color patterns in the common leopard frog.

Background of the Problem

Two color phases, the non-spotted leopard frog and the mottled leopard frog, differ markedly in dorsal pattern from the common leopard frog, *Rana pipiens* (Figure 17). The non-spotted leopard frog, originally described by Weed[13] in 1922 as a new species, *Rana burnsi*, lacks the dorsal spots that characterize the common leopard frog. The mottled leopard frog, also designated by Weed[13] as a new species, *Rana kandiyohi*, is distinguished from the common leopard frog by black vermiculate mottling in the interspaces between the dorsal spots. Weed contended that the differences in color pattern are so striking and constant as to warrant a new taxonomic status for each color phase. However, Moore[6] in 1942 demonstrated that the non-spotted leopard frog differs from the common leopard frog simply by one dominant gene that affects the color pattern. (The gene for the non-spotted color pattern is designated B; the recessive common-spotted pattern allele is designated B^+.) Volpe[12] in 1955 showed that the color pattern of the mottled leopard frog is also the expression of a single dominant gene that influences pigmentation. (The dominant mottled color pattern gene is designated K; the common-spotted pattern gene is designated K^+.) In light of

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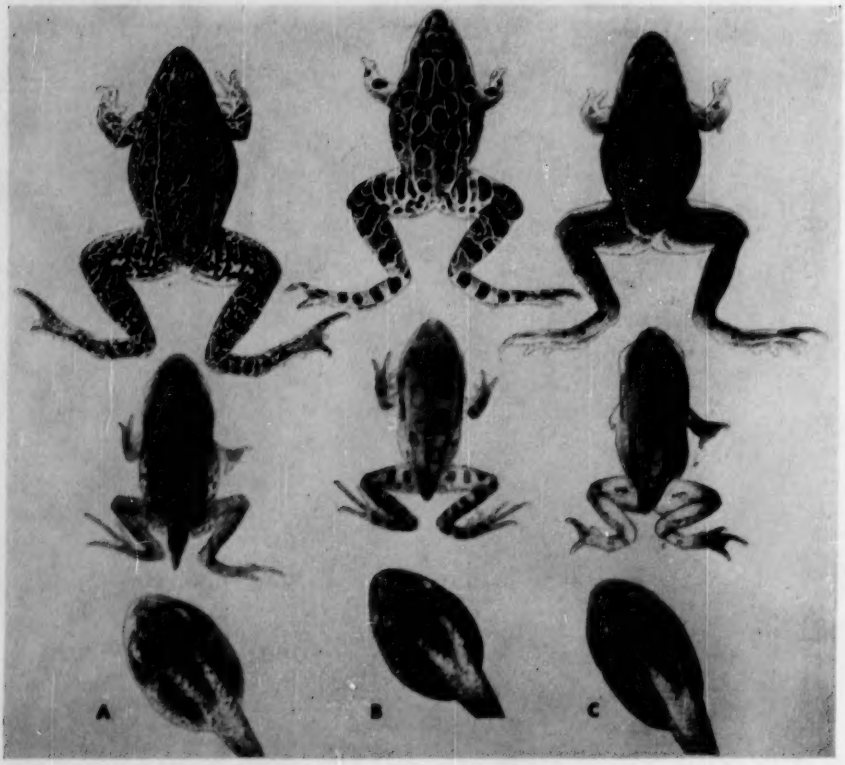


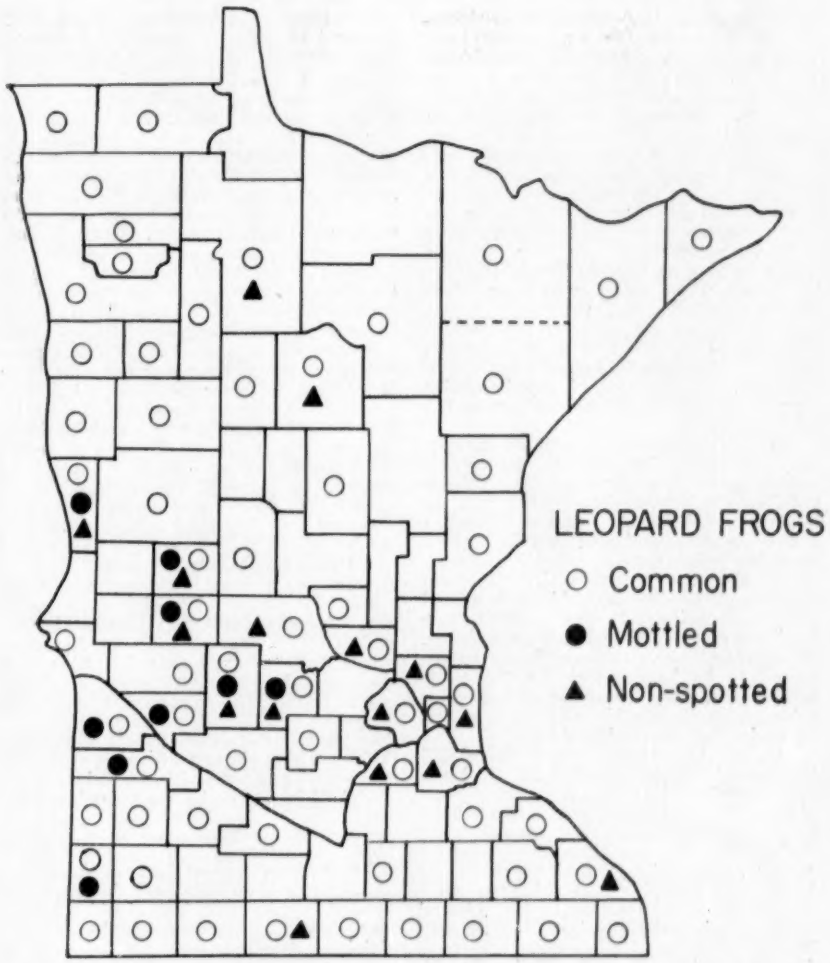
LIFE CYCLE OF THREE COLOR TYPES

Figure 17

Three stages (larval, metamorphosis, and adult) in the life history of *A—Rana pipiens* [Kandiyohi dominant mutant]; *B—Rana pipiens;* and *C—Rana pipiens* [burnsi dominant mutant].

these findings, neither color phase warranted the status of species, and subsequently both were reduced to synonymy with *Rana pipiens.* The non-spotted leopard frog and the mottled leopard frog are now referred to taxonomically as *Rana pipiens* [burnsi dominant mutant] and *Rana pipiens* [kandiyohi dominant mutant] respectively.

Knowledge of the distribution of each mutant type in natural populations is incomplete.[1,10,13,14] Localities from which *Rana pipiens* [kandiyohi dominant mutant] have been recorded are, with one exception, in the southwestern portion of Minnesota. The exceptional record is from the southeastern part of South Dakota close to the border of Minnesota. The center of distribution of *Rana* pipiens [burnsi dominant mutant] is also southern Minnesota, with peripheral populations in the adjacent states of Iowa, South Dakota, and Wisconsin. When the locality records for each mutant type are plotted from Breckenridge's[1] distributional data on leopard frogs in Minnesota, it is noted (Figure 18) that the non-spotted leopard frog always coexists with the common leopard frog, the mottled leopard frog always coexists with the common leopard frog, and all three types have been found



DISTRIBUTION OF LEOPARD FROGS IN MINNESOTA

Figure 18

The distribution marks of the common, mottled and non-spotted leopard frogs signify that they have been found in the counties indicated. Distribution map adopted from Breckenridge.[1]

together in several localities. The restricted and almost similar distribution of the mutant types, their coexistence with the common leopard frog in several localities, and the fact that the differences between the three forms reflect simply changes in the same part of the organisms suggested prior to experimentation that the three color patterns are intimately allied genetically.

Breeding Experiments

The three forms have been crossed in all possible ways by artificial fertilization. In the following discussion, the common leopard frog, the mottled leopard frog, and the non-spotted leopard frog are referred to simply as pipiens, kandiyohi, and burnsi respectively. Kandiyohi and pipiens were previously crossed (Volpe[12]).

A heterozygous kandiyohi bred with a pipiens (the double recessive) yields a ratio of 1 kandiyohi : 1 pipiens. As Moore[6] demonstrated that burnsi differs from pipiens by a single dominant gene, burnsi and pipiens were crossed only once. The burnsi individual proved to be heterozygous for the dominant color-pattern gene, yielding a 1:1 ratio when crossed with pipiens. Breeding experiments were completed by crossing the two mutant types, kandiyohi and burnsi. The series of crosses involving the two mutant frogs form the basis of the present report.

Five series of artificial fertilizations involving kandiyohi and burnsi were made. Each series, designated A, B, C, etc. (Table I), consisted of the following crosses: kandiyohi ♀ × kandiyohi ♂, kandiyohi ♀ × burnsi ♂, burnsi ♀ × kandiyohi ♂, and burnsi ♀ × burnsi ♂. The methods of experimentation were the same as those employed in an earlier work (Volpe[12]).

The results of the experiments are summarized in Table I. The adult color pattern first appears at the time of metamorphosis (Figure 17). The metamorphosed young from the kandiyohi × kandiyohi crosses showed a sharply defined segregation into the mottled kandiyohi color pattern and the common-spotted pipiens color pattern. A total of 220 transformed in the kandiyohi × kandiyohi crosses, of which 171 (77.7 percent) were kandiyohi and 49 (22.3 percent) were pipiens. All kandiyohi adults employed in the crosses proved to be heterozygous for the color-pattern gene. (The probability of uncovering a homozygous kandiyohi individual in natural populations will be considered later.)

The burnsi × burnsi crosses yielded 219 transformed tadpoles, of which 165 (75.3 percent) were burnsi and 54 (24.7 percent) were pipiens. Here also, all burnsi adults utilized in the experiments were heterozygous.

Combining the results of the reciprocal crosses (kandiyohi × burnsi and burnsi × kandiyohi), a total of 502 offspring segregated into 245 kandiyohi (48.8 percent), 133 burnsi (26.5 percent) and 124 pipiens (24.7 percent). The actual segregating population recovered from the reciprocal crosses fits by the chi-square test an expected ratio of 2 kandiyohi: 1 burnsi: 1 pipiens.

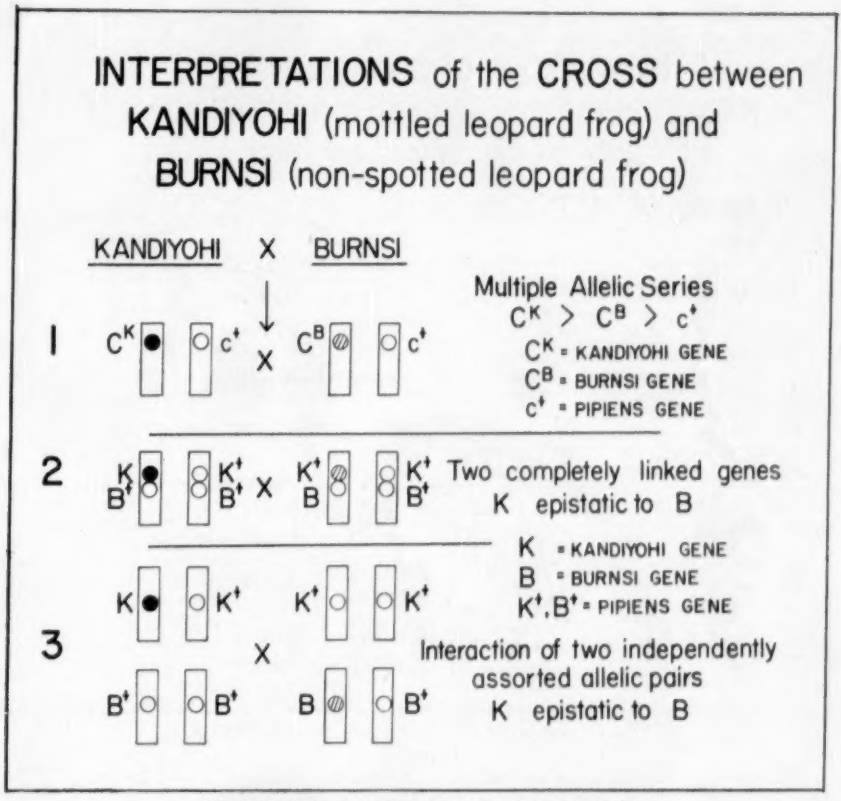
The embryos resulting from the reciprocal crosses (kandiyohi × burnsi and burnsi × kandiyohi) in Experiments D and E were analyzed at various stages of development to determine the first point in development where differences in pigmentation could be discerned. The pattern of epidermal melanophores is similar in the three kinds of tadpoles, but the amount and distribution of the subepidermal melanophores are so strikingly different as to constitute a valid criterion for separating the tadpoles into three categories. Figure 17 illustrates the appearance of the three kinds of tadpoles; the "lightly pigmented" tadpole transforms into a burnsi, the "weakly pigmented" tadpole gives rise to a pipiens and the "heavily pigmented" tadpole undergoes metamorphosis into a kandiyohi. The pigmentation differences are traceable back to Larval Stage X (defined in Taylor and Kollros[11] as that larval period in which the margin of the hind-limb bud is indented between the digit primordia). A total of 112 tadpoles from the reciprocal crosses in Experiments D and E were classified at Stage X on the basis of the amount of subepidermal melanophores and separated into individual culture dishes. Of the 52 tadpoles that were originally classified at Stage X as "heavily pigmented," 48 (92.3 percent) transformed into kandiyohi adults and 4 (7.7 percent) into pipiens adults. Of the 31 tadpoles classified as "weakly pigmented," 26 (83.9 percent) gave rise to pipiens adults, 2 (6.4 percent) gave rise to kandiyohi adults, and 3 (9.7 percent) gave rise to burnsi adults. Of the 29 tadpoles classified as "lightly pigmented," 26 (89.7 percent) transformed into burnsi and 3 (10.3 percent) into pipiens. Thus, the adult pigment differences are heralded in the larval stage. The exceptions can be explained in part by incorrect classification.

Discussion

In reciprocal crosses between the mottled leopard frog (kandiyohi) and the non-spotted leopard frog (burnsi), both of which are mutant forms of the common leopard frog (pipiens), two facts were revealed: first, not only were the mottled kandiyohi color pattern and the non-spotted burnsi color pattern recovered, but the common-spotted pipiens color

TABLE I. Results of crosses between Kandiyohi and Burnsi (K = Kandiyohi; B = Burnsi; P = Pipiens)

Experiment	K♀ × K♂		K♀ × B♂			B♀ × K♂			B♀ × B♂	
	K	*P*	*K*	*B*	*P*	*K*	*B*	*P*	*B*	*P*
A	40	14	24	10	14	18	13	9	34	11
B	31	6	26	16	11	29	14	10	42	9
C	36	10	23	12	13	30	11	13	33	13
D	28	8	24	15	17	24	16	12	27	12
E	36	11	20	11	12	27	15	13	29	6
Total	171	49	117	64	67	128	69	57	165	54
Expected	165	55	124	62	62	127	63.5	63.5	162	57
Chi Square	0.87		0.86			0.68			0.22	
Probability	0.35		0.65			0.71			0.64	

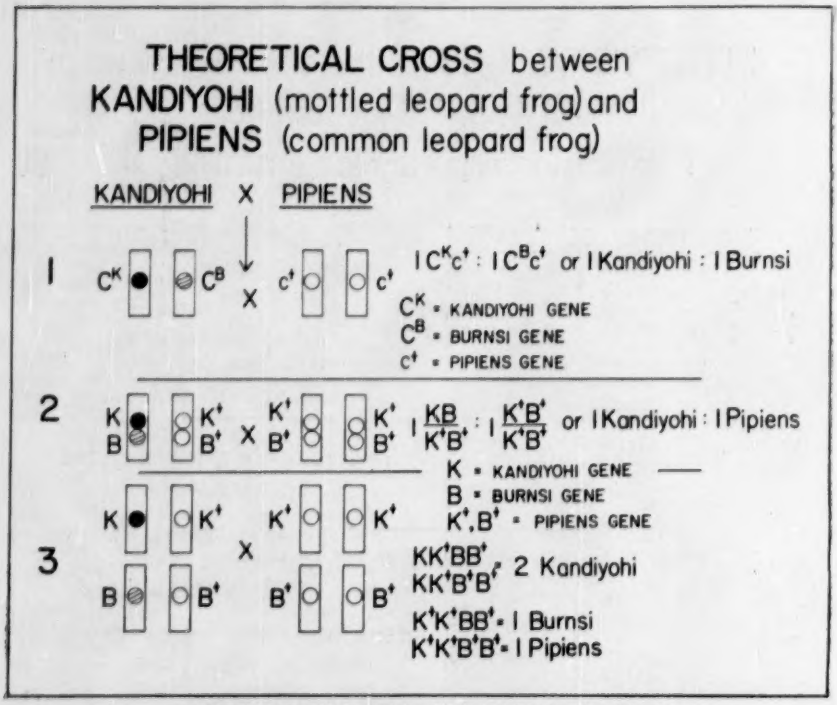


CROSSES BETWEEN TWO MUTANT FROGS
Figure 19

Crosses between the mottled leopard frog (kandiyohi) and the non-spotted leopard frog (burnsi), both of which are mutant forms of the common leopard frog (pipiens), yielded offspring which segregated into a ratio of 2 kandiyohi: 1 burnsi : 1 pipiens. Three possible interpretations of the 2:1:1 ratio are represented.

pattern was obtained as well; and secondly, the offspring segregated into a ratio of 2 kandiyohi: 1 burnsi: 1 pipiens. Three possible interpretations of the 2:1:1 ratio are diagrammed in Figure 19. First, the three color pattern genes may represent a multiple allelic series, with the kandiyohi gene (C^K) dominant over both the burnsi (C^B) and pipiens ($c+$) alleles, and the burnsi gene dominant over the pipiens allele. A second explanation involves two completely linked genes, with the kandiyohi gene (K) masking the expression of the burnsi (B). The first explanation is simpler if one assumes that multiple alleles affect principally the same region of the organism in different phenotypes and that in linkage widely different parts of the organism are affected in the different phenotypes. However, this distinction is probably not valid, particularly in light of the discovery of pseudoallelism (even the classical series of multiple alleles at the white eye-color locus in *Drosophila* has been demonstrated to involve two different, although closely adjacent, loci[5]). A third explanation involves an interaction of two independently located pairs of genes, with the kandiyohi gene (K) epistatic to the burnsi gene (B).

The correct interpretation can be resolved by backcrossing experiments, but this is dependent, regrettably, upon the ease with which sexually mature frogs can be reared in the laboratory. It is also possible to differentiate among the alternatives if one were to find in natural populations a kandiyohi individual carrying the burnsi gene. Figure 20, which is self-explanatory, represents the differentiating ratios that would result if a kandiyohi


THEORETICAL CROSS between
KANDIYOHI (mottled leopard frog) and
PIPIENS (common leopard frog)
KANDIYOHI X PIPIENS
1
1 C^Kc^+ : 1 C^Bc^+ or 1 Kandiyohi : 1 Burnsi
C^K = KANDIYOHI GENE
C^B = BURNSI GENE
c^+ = PIPIENS GENE
2
1 $\frac{KB}{K^+B^+}$: 1 $\frac{K^+B^+}{K^+B^+}$ or 1 Kandiyohi : 1 Pipiens
K = KANDIYOHI GENE
B = BURNSI GENE
K^+,B^+ = PIPIENS GENE
3
KK^+BB^+
$KK^+B^+B^+$ = 2 Kandiyohi
$K^+K^+BB^+$ = 1 Burnsi
$K^+K^+B^+B^+$ = 1 Pipiens

THREE POSSIBLE MODES OF INHERITANCE

Figure 20

Three modes of inheritance can be postulated from the results of crosses involving kandiyohi, burnsi, and pipiens (see text and Figure 19). The inheritance scheme could be clarified if a kandiyohi individual of a specific genotype could be found and mated to a pipiens individual. The differentiating ratios resulting from such a cross are shown at the right.

individual harboring the burnsi gene (specifically, C^KC^B or $KB/K+B+$ or $KK+BB+$, depending upon the interpretation) were to be crossed to a pipiens individual ($c+c+$ or $K+K+/B+B+$ or $K+K+\ B+B+$).

Knowledge of the relative proporitions of kandiyohi, burnsi, and pipiens individuals in wild populations can be used to ascertain the probable gene distribution and the chance frequencies of matings which would yield a kandiyohi individual of the desired genotype. In localities in Minnesota where burnsi and pipiens have been collected together, Mr. Steinhilber (professional collector) estimates a ratio of one burnsi to 99 pipiens. In addition, he estimates that collections of kandiyohi and pipiens from habitats in which both occur contain one percent kandiyohi. Unfortunately, no information could be obtained regarding the relative proportions of the three kinds of frogs in those localities where all three forms occur together. Treating each approximation separately, and assuming that the habitats from which the leopard frogs have been collected support a population large enough to allow random breeding, the Hardy-Weinberg formula may be used to estimate the relative frequencies of homozygous kandiyohi (C^KC^K or $K-/K-$ or $KK--$),* heterozygous kandiyohi (C^Kc+ or $K-/K+-$ or $KK+--$) and homozygous pipiens ($c+c+$ or $K+-/K+-$ or $K+K+----$). Similarly, the relative frequencies of homozygous burnsi (C^BC^B or $-B/-B$ or $--BB$), heterozygous burnsi (C^Bc+ or $-B/-B+$ or $--BB+$) and homozygous pipiens ($c+c+$ or $-B+/-B+$ or $--B+B+$) may be predicted. Thus, in a population consisting of one percent kandiyohi, the equilibrium formula of $q^2\ KK : 2q\ (1-q)\ KK+ : (1-q)^2\ K+K+$ reads 0.000025 KK : 0.009950 $KK+$: 0.99 $K+K+$. On the basis of 40,000 individuals, one out of the 40,000 would

*Neglect (or assume homozygosity for) the possible second pair of genes.

be homozygous kandiyohi and 398 out of the 40,000 would be heterozygous kandiyohi individuals. It is not remarkable then that all the kandiyohi individuals used in the breeding experiments proved to be heterozygous. Calculations for the relative frequencies of homozygous and heterozygous burnsi individuals are numerically identical. Thus, assuming a chance meeting of a kandiyohi and a burnsi, the probabilities of matings between a homozygous kandiyohi and a homozygous burnsi, a heterozygous kandiyohi and a heterozygous burnsi, and a heterozygous kandiyohi and a homozyogus burnsi would be $1/16 \times 10^8$, $1/10^4$, and $1/4 \times 10^6$ respectively. Indeed, the probability of finding in nature a kandiyohi individual carrying a burnsi gene is small. The best explanation for the 2:1:1 ratio obtained experimentally awaits the design of a successful technique for rearing sexually mature frogs in the laboratory.

Summary

Collections of the common leopard frog, *Rana pipiens*, from the north central states contain two mutant types, the mottled leopard frog and the non-spotted leopard frog. It has been demonstrated previously that the mottled leopard frog differs from the common leopard frog simply by one dominant gene that influences pigmentation. The non-spotted leopard frog is also a single dominant color-pattern mutant of the common leopard frog. In the present study, the results of breeding experiments have indicated that the three color patterns, the mottled pattern, the non-spotted pattern, and the common-spotted pattern may be determined by a multiple allelic series with dominance in the order given. Crosses of the mottled leopard frog with the non-spotted leopard frog yield a phenotypic segregation into a statistically significant ratio of 2 mottled leopard frogs : 1 non-spotted leopard frog : 1 common leopard frog. Other genetical interpretations of the data are discussed.

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WHITE MUTANTS OF *SERRATIA MARCESCENS*

A Study of Physiological and Morphological Characteristics

Micheline M. Mathews*

Several types of color variants of *Serratia marcescens* have been described.[2,3,5] These have been found to occur naturally in aging cultures and have been induced by different means. In previous studies of these forms, the morphological and physiological characteristics of the mutant forms seem not to have been emphasized. This paper is a study of the morphological and physiological characteristics and the reactions to antibiotics of white mutants of *Serratia marcescens*.

Materials and Methods

The strain of *Serratia marcescens* used in this study was one purchased from the Carolina Biological Supply House. It produces stable red pigmented colonies. The bacteria were cultured in

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glycerol-peptone medium.[5]

Mutants were obtained by irradiation and by growing the bacteria in autoclaved broth cultures of other organisms as described by Bellini.[1] In the first method, the bacteria were irradiated in open dishes in darkness at a distance of 20 inches from a Westinghouse W1 Type Sb Sterilamp for thirty seconds, and the irradiated suspension was then plated out. In the second method, 24 and 48 hour broth cultures of *Escherichia coli* and *Bacillus subtilis* were autoclaved and then inoculated with the *Serratia*. Plate counts were done on the first, third, and fifth days of incubation. In both methods, plating was done in glycerol-peptone agar.

The presence of white mutants was determined by inspection, and the ratio of white to red colonies was determined. White colonies were isolated, and Gram stains and subcultures were made. The mutants were grown in triple sugar iron agar, starch agar, nitrate agar, and tryptone broth. The action of Penicillin and Streptomycin on these forms was determined by the serial dilution method, and the action of antibiotic-producing bacteria was tested as described in Waksman.[7] The rate of respiration of the normal and mutant forms was determined in a Barcroft respirometer using standard manometric techniques.[4, 6]

Results and Discussion

White variants of *Serratia marcescens* have been obtained by the methods given above. These forms have been found to breed true, after numerous subculturings, for a period of about six months. None have ever reverted to the red form. Table I shows the ratio of white to red forms. Since the rate of mutant production is many times greater than the normal rate of occurrence, it may be that there is a mutagenic factor present in the autoclaved culture. Whether this factor is something in the killed bacteria, some waste product of their metabolism or a disintegration product is not known at this time. The rate of mutation by irradiation was not as great as that given in Table I.

TABLE I. Ratio of white to red colonies obtained when *Serratia marcescens* was grown in autoclaved broth cultures

Autoclaved broth culture	Ratio of white to red colonies 1 day	3 days	5 days
None	1:20,000		
Bacillus subtilis (24 hr. culture)	1:40	1:40	1:20
Bacillus subtilis (48 hr. culture)	1:33	1:10	1:10
Escherichia coli (24 hr. culture)	1:100	1:50	——
Escherichia coli (48 hr. culture)	1:50	1:20	1:20

Upon detailed examination under oil-immersion of the Gram stains of the white variants, some differences were seen in the parent and mutant cells. Those of the variants appeared to be longer, and some tended to form chains. Some were larger than usual and contained granules. Both mutants and parent forms were Gram-negative. This difference in microscopic appearance coincides with the findings of Bellini with mutants of *Sarcina lutea*.[1]

The colony appearance of both parent and mutant forms are similar, the only difference being in the pigmentation. The reactions in the various physiological test media are the same for both forms. This fact also coincides with Bellini's findings. The respirometer studies show a slight increase in the respiration rate of the white variant.

The white forms were found to be somewhat more resistant to Streptomycin, but not any more resistant to Penicillin and the antibiotic-producing bacteria than was the parent type. Similar results were obtained in a previous study of *Sarcina lutea* in which mutants obtained were not only more resistant to Streptomycin, but also to antibiotic-producing bacteria.

Summary

A mutant form of *Serratia marcescens* has been obtained. This differs somewhat in microscopic appearance from the parent type, but colonial characteristics (except for pigment) and reactions in various test media are the same. The mutant is slightly more resistant to Streptomycin and has a somewhat faster rate of respiration. It has bred true for a period of about six months.

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FIRST INTERNATIONAL CONGRESS OF HUMAN GENETICS

Copenhagen; August 1-6, 1956

ACCORDING to the most recent information received from the congress office (Tagensvej 14, Copenhagen N.), satisfactory progress has been made in the organizational arrangements for the first international congress of scientific branches concerned with the clinical and experimental aspects of human genetics. Owing to the most thorough planning of Prof. Tage Kemp and his staff, the preparatory work has reached a point where the prediction of a successful meeting is no longer a matter of speculation.

Since the active participation of leading experts from many countries has long been assured, it is a particular pleasure to report that the size of the American contingent will be respectable. Nearly fifty geneticists from the United States have paid their enrollment fee since April 1, 1956.

The scientific meetings and exhibitions will be held at the Institute of Medical Anatomy, 63 Norre Alle, Copenhagen North, with a maximum time limit of 15 minutes for each paper. Presentation of papers in the English language has been recommended, although French and German will also be official languages of the congress. Simultaneous interpretation has been arranged for all important sessions.

The topics of the plenary sessions (forenoon) will include the following: Mutation in man and Radiation genetics and its human implications; Comparative and experimental pathology in relation to human genetics and Genetics and medical research; Methods in human genetics; Biochemical genetics; and Social applications of human genetics and Epidemiological control of hereditary diseases (August 6). Sectional afternoon meetings will deal with specialized genetic problems in internal medicine; Etiology of cancer; Psychiatry, neurology, and psychology; Biochemistry; Anthropology; Blood groups; Ophthalmology; Odontology; and Population genetics. An excursion to Elsinore is planned on Sunday, August 5.

Final arrangements for the social part of the congress have yet to be completed. In addition to an official banquet (probably on August 4), there will be a City Hall reception and several other festivities. Formal dress will be optional.

Although the hotel situation may prove to be difficult, the congress office will accept applications for living accommodations (Form D) until June 20, 1956. Tours before and after the congress may be arranged through Dantourist, the official travel agency.

Committee on International Meetings,
American Society of Human Genetics

"DEAFMUTISM" IN TWO PAIRS OF IDENTICAL TWINS

RICHARD H. POST AND LOUISE A. HOPKINS*

LUCHSINGER and Hanhart[3] have described considerable differences in the hearing patterns of three pairs of monozygotic twins with congenital hereditary (nerve) deafness, sometimes called "sporadic" or "recessive deafmutism." One of each pair was profoundly deaf and the other only partially deaf. The cases demonstrate a wide variation in penetrance or expressivity in the homozygote, of a relatively familiar gene or genes.

Gedda, Maltarello and Rédeky[2] published audiograms of five pairs of twins described as congenital recessive deafmutes, four of them monozygotic and the fifth probably so. Each pair showed a similarity in their hearing curves, some being practically identical.

Summary of Present Cases

Two pairs of monozygotic twins are described, one pair with strong resemblances in audiometric patterns, histories of congenital "deafmutism" and a sister who has a similar history; the other pair with marked differences in audiometric patterns and no congenitally deaf relative.

The first pair is believed to have hereditary congenital nerve deafness, and thus provides another case of close similarity in gene expression. The deafness of the second pair is believed to be either "acquired" or, if hereditary, of a different origin and form.

The pairs will be designated I and II, respectively. All four, also the deaf sister of Pair I, are pupils at the Clarke School for the Deaf in Northampton, Massachusetts.

The close resemblance of the audiograms of Pair I is striking (Table I). While the audiogram of II B resembles those of Pair I, that of II A is distinctly different and indicates that she has some degree of usable hearing. This is apparent from her more normal speech.

Family Histories

Pair I have a congenitally deaf sister with no record of sickness in infancy. She has been given adequate medical attention, particularly since admission to the Clarke School in 1941, at the age of five. No other relative is known to have had hearing difficulties except two great-aunts (their father's mother's sisters) who had abscessed ears during childhood and commenced to lose hearing in their late twenties. One was deaf at 28 and the other presumably at a similar age. Both wore hearing aids and died in their sixties, one of a stroke. They had four sisters, who produced 15 children, and three brothers, who produced 10 children—all with normal hearing. Other normally hearing relatives include the two parents, four grandparents, mother's three sibs with their three children, father's sister with two children, father's father's three sibs with five children and seven grandchildren (as of 1948).

Although the deafness of two great-aunts suggests that a late-appearang form of recessive deafness may "run in the family," the fact that it is limited to two cases, added to the fact that the twins' sister's deafness is apparently of the same type as their own, points to congenital hereditary deafness.

Pair II have a first cousin, their father's sister's daughter, who became deaf in the left ear after a radical mastoid operation. There is no other deafness in the family, which is large: parents and grandparents, nine aunts and uncles and their 15 children on the father's side; on the mother's side eight aunts and uncles, their seven children,

TABLE I. Audiograms, showing decibel losses at seven standard frequencies from 128 d.v. per sec. to 8192 d.v. per sec.

	IA		IB		IIA		IIB	
Frequency	R	L	R	L	R	L	R	L
128	60	65	60	60	60	55	70	60
256	70	70	65	65	70	55	75	70
512	85	75	80	80	75	70	90	80
1024	95	85	95	95	75	70	100	65
2048	95	90	95	105	65	65	105	100
4096	100	100	115	110	60	65	105	100
8192	----	----	----	----	55	55	----	----

*Institute of Human Variation, Columbia University, New York, N. Y., and The Clarke School for the Deaf, Northampton, Massachusetts, respectively.

28 first cousins of the mother and their children (as of 1950).

Zygosity Diagnosis

Intra-pair resemblances of finger patterns and ridge counts are striking (Table II). Important pattern differences are limited to index fingers, considering the reversed symmetry in the ring fingers of pair II as a resemblance. Ridge count totals are relatively close: IA=170, IB=167, a difference of 3; IA=163; IIB=141, a difference of 22. Such a small difference in pair I is strong indication of monozygosity[1]. Pair II would be closer but for the differences in thumb and fourth fingers of the left hands.

Pair I (Table IV and V), are so similar that most observers would probably confuse them at first were it not that B wears glasses, marks of which are apparent when removed.

They appear almost indistinguishable in the following traits: peculiar yellowish color and general pattern of teeth; head hair (light brown, moderately coarse in texture, with low waves); eyebrow outline or form; hair pattern on fingers, hands and forearms; eye color, iris texture, form of palpebral opening and eyelids; details in form of cheeks, nose, lips, chin, ears; slight freckling pattern; skin color in various parts of face, neck and arms.

Very slight differences could be noted, but only upon careful inspection, in the following: profile of forehead more bulbous in A, flatter in B; lip prognathism greater in B; upper lip philtrum more sharply defined in A; right forearm hair less abundant in A; right little finger hair less abundant in B. These differences are all quite minor.

Pair II are easily distinguished by the larger size of A, as shown by most of the body measurements, (Table V). Her greater facial breadth is reflected by a bizygomatic measurement of 129 millimeters, contrasted with 125 for B. The lower jaw of A is broader by 2 millimeters and there is a small but clearly marked hiatus or gap between the upper middle incisors. No other difference in the teeth could be detected, even the degree of crowdedness appearing identical.

Pair II appear almost indistinguishable for every feature noted above for Pair I. The only differences noted, other than that of size, are that A has a straighter nasal bridge, B a slight convexity; A's nose tip is slightly smaller; A's lip membranes are slightly more conspicuous or "thicker."

TABLE II. Finger patterns and ridge counts. (DW =double whorl; CPUL=central pocket ulnar loop).

		Thumb	Index	Middle	Ring	Little
IA	R	UL	W	UL	UL	UL
			8	15	16	18
		24	d=1			
	L	UL	DW	UL	UL	W
		27	d=8	15	16	d=9
IB	R	UL	UL	UL	UL	UL
		22	16	13	15	15
	L	UL	RL	UL	UL	W
						7
		22	15	14	17	d=11
IIA	R	DW	W	UL	W	UL
		13	13	12	14	12
		d=15	d=1		d=2	
	L	CPUL	RL	W	CPUL	UL
		14	14	12	8	16
		d=10		d=3	d=6	
IIB	R	DW	UL	UL	CPUL	UL
		14	11	8	5	14
		d=14			1=8	
	L	W	RL	W	"	UL
		19	8	9	"	17
		d=1		d=1	d=1	

TWIN PAIRS I AND II

Figure 21

Pair I above; A left, B right. The audiograms of Pair I show a striking resemblance. Pair II, below; A right, and B left. The audiograms of Pair II are distinctly different and indicate that IIA has some degree of usable hearing.

Medical Observations

Pair I were born March 17, 1942 at full term without complications. They were not breast fed. They are believed to have been born deaf, and there is no history of infection prior to the discovery of deafness. No important differences occur in the histories of child-

TABLE III. Five blood groups.

I A	A	MN	Cc	D	ee
I B	A	MN	Cc	D	ee
II A	O	MN	cc	D	E
II B	O	MN	cc	D	E

hood sicknesses. Both have had five molar inlays.

The only important difference between them is a mild internal strabismus of the left eye of B, noted at age four and fully corrected by glasses in 1955, with prescription; Right eye -1.25 $-.50 \times 180°$; Left eye -1.00 $-1.00 \times 180°$.

Of minor importance is a pilonidal depression about one-half inch in diameter on the right side of the base of the spine of A, which is congenital. B has lost her upper right central incisor, replaced in 1955.

At age 12 years five months their educational ages were both six years two months; grade equivalent, 2-8.

Pair II were born June 18, 1942 at eight months' term and incubated for three weeks. A weighed 65 ounces, B 60. They were not breast fed. A sat up at six months, B at 6½ months; A teethed at eight months, B at nine months; neither had convulsions; A walked at 11 months, B at 11½ months. Deafness was noted in both at 15 months but is believed to have been congenital. Neither had any illness during infancy nor has either had many colds.

B had a skin cyst in the midline of the suprasternal notch. First reported in 1948, it was removed in 1950 when three-quarters of an inch in diameter. A has a small hole on the left side of her neck.

Both twins had prominent or "winged" scapulae in 1953, B again in 1954, but this condition was not noted in 1955. Both had heat murmurs each year from 1948 through 1949 but not before nor since.

As shown by the audiograms, (Table I), A has some useful hearing. She learned to talk by the age of 2½ but her speech was very imperfect. B has no functional hearing.

Both twins had several episodes of otitis media before their tonsils and adenoids were removed at age three. B's attacks commenced at 29 months when her right ear discharged but there is no history of the left. A's attacks were less severe. This infection was not considered responsible for deafness by the Massachusetts General Hospital in a report of 1946.

Both had chicken pox at three and measles at four, B a little more severely than A.

At age 12 years three months A (the twin with some hearing) had an educational age of 10 years four months, B of eight years 10 months. Their grade equivalents were 4.1 and 3.2 respectively. A has always seemed to the mother as being more happy, cooperative, dependable for errands and easier to work with than B. The mother explains this difference as being due to her superiority in hearing.

TABLE IV. Height in inches and weight in pounds, in September of each year

	I-A		I-B		II-A		II-B	
	Ht.	Wt.	Ht.	Wt.	Ht.	Wt.	Ht.	Wt.
1946	38	33	38	33	—	—	—	—
1947	41	38	41	38	41	33	40	32
1948	44	42	44	43	43	37	42	36
1949	46	46	46	46	45	43	44	40
1950	48	51	48	51	47	50	46	48
1951	50	58	50	58	49	55	48	57
1952	52	63	53	65	51	60	50	58
1953	55	77	56	74	54	76	54	68
1954	58	89	58	85	58	94	57	82

Discussion and Conclusion

There is little doubt that both pairs of twins are monozygotic, and it seems quite likely that Pair I has congenital hereditary nerve deafness, since a sister has a similar history and since there is no aetiological explanation except that of a genetic nature. The cause of deafness of Pair II cannot be explained. No other case of congenital deafness appears in their fairly large fraternity, and twin A has considerably more hearing than B. Their deafness may have some connection with their premature birth. On the other hand it is possible that they manifest a form of "recessive deafmutism" with a wide degree of gene expression.

The close similarity of the audiograms of Pair I supports the finding of Gedda *et al*[2], that gene expression may be highly constant in congenital hereditary deafness.

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TABLE V. Anthropometric comparisons, May 1955

	I A	I B	II A	II B
Sitting height	1772	1785	1808	1795
Shoulder breadth	322	314	302	307
Head length	188	197	178	176
Head breadth	146	143	141	142
Face height	109	110	122	122
Jaw breadth	99	99	95	93
Nose length	46	47	53	54
Nose breadth	32	32	33	32
Ear length, L	56	57	59	58
Ear length, R	60	60	58	57
Ear breadth, L	32	35	37	35
Ear breadth, R	32	35	34	35
Foot length, L	228	233	241	176
Foot length, R	225	233	243	176
Foot breadth, L	84	83	84	82
Foot breadth, R	83	83	84	81

BASIC NUMBERS IN THE POLYPLOIDY OF *SACCHARUM*

ICHIZO NISHIYAMA*

SINCE the discovery of an extensive polyploid series (n = 24, 27, 28, 30, 32, 36, 40, 48, 50, 56, 60, 64) in the genus *Saccharum*, this polyploidy has been discussed by several authors from a point of view of different basic numbers; *e.g.*, 10 (Bremer[5,6]) eight (Dutt and Rao[13]), 10 and 12 (Darlington and Janaki-Ammal[12]), and five (Parthasarathy,[28] Raghavan[32]). The disagreement in the basic number might be due to the fact that (1) no diploid plant ($2x$) in *Saccharum* has yet been found, and (2) no experimental study has been designed for genome analysis in *Saccharum* and its relatives. Knowledge of the evolution of polyploidy in *Saccharum* is very important. Not only does such knowledge have fundamental significance, but it might also play an important role in the breeding of sugarcanes.

About 10 years ago the author and his colleagues began to study the cytogenetics of *Saccharum* and its allies under the direction of Dr. H. Kihara. After a short time the work was suspended. The few papers concerning this work were published in Japanese.

The author is pleased to have the opportunity to continue the study of sugarcanes. The present work was carried out at the Experiment Station of the Hawaiian Sugar Planters' Association and in the U. S. Department of Agriculture. The conclusion obtained by the author presents a new aspect on the genomic constitution of the polyploidy in *Saccharum*. A complete survey can not be given here. Some results will be mentioned which, it is hoped, will suffice.

Material and Methods

All varieties of sugarcanes and all interspecific hybrids used in this work were grown at the Experiment Station of the H.S.P.A. in Hawaii. P.M.C.'s were fixed in Carnoy's fixative (3:1) for 24 hours or more. Materials were collected during October-December, 1954.

Whole anthers were stained with aceto-carmine for about one hour following a mordant pretreatment (four per cent aqueous solution of iron alum) and were squashed on clean slides.

Observations

Special attention was paid to the meiotic pairing of chromosomes in metaphase I of P.M.C.'s. That is, the number of bivalents and univalents was carefully counted. Sometimes trivalents and quadrivalents were observed. These multivalents were usually formed through weak conjugation between two bivalents or between one bivalent and one univalent. Owing to loose connections, it was often difficult to determine the formation of complex pairing.

1. *S. officinarum* (n=40) and *robustum* (n=40) × *spontaneum* Coimbatore (n=32)

Included in Table I are the results of cytological observations on four hybrids between certain varieties (n=40) of *S. officinarum* and *S. spontaneum* Coimbatore (n=32).

*Contribution No. 24 from the Laboratory of Applied Genetics, Research Institute for Food Science, Kyoto University, Kyoto, Japan. The author wishes to express his sincere thanks to the Yale-Bishop Museum Foundation Fellowship for the financial help with which this work was carried out. The author is much indebted to Dr. A. J. Mangelsdorf of the Experiment Station, Hawaiian Sugar Planters' Association, for many valuable suggestions and for providing him with materials used during the course of the research. It is also a pleasure for the author to acknowledge the help and encouragement of Dr. E. W. Sinnott, Yale University; Dr. J. N. Warner and Mr. R. Urata, Experiment Station, Hawaiian Sugar Planters' Association; as well as Dr. H. M. Tysdal, Mr. C. O. Grassl, Dr. I. E. Stokes, and Dr. S. Price, U. S. Department of Agriculture, Beltsville, Maryland.

METAPHASE I CHROMOSOMES

Figure 22

A—Chromosomes in metaphase I of *Saccharmum officinarum* (H 109, $n = 40$) × *S. spontaneum* Coimbatore ($n = 32$). $50_{II} + 3_I = 103$. *B*—Chromosomes in metaphase I of *S. officinarum* (D 908) × *S. spontaneum* Coimbatore ($n = 32$). $55_{II} + 2_I = 112$.

(1) H 109 (n=40) × *S. spontaneum* Coimb (n=32)—H53-9457.

In metaphase I of this hybrid, the author counted clearly $50_{II}+3_I$ (Figure 22*A*) and $1_{III}+49_{II}+2_I$ twice in each case. In many other nuclear plates there were observed no abnormalities in chromosome behavior, excepting zero to five lagging univalents in anaphase I. Five laggards, the maximum number so far observed, were found in two cells. With some exceptions, lagging univalents were longitudinally divided and their halves moved to the opposite poles. Usually univalents were included in the daughter nuclei, but sometimes they formed micronuclei.

From counts in metaphase I, the somatic chromosome number was found to be $2n$=103. This number is 31 chromosomes in excess of the expected, $2n$=72 (40+32). The unexpected increase of the chromosome number will be discussed later.

(2) D 908 (n=40) × *S. spontaneum* Coimb. (n=32)—H 51-9035

This hybrid is from a cross similar to the one given above. However, the author found $55_{II}+2_I$ in four metaphase I plates (Figure 22*B*) and 56_{II} in one plate. In this hybrid two to three of the bivalents appeared to show a weak pairing. The number of lagging univalents was usually zero to two or sometimes three to four in ana-telophase I. From these results it can be said that this hybrid has 40 chromosomes in excess of the expected diploid number ($2n$=72).

(3) Co. 205

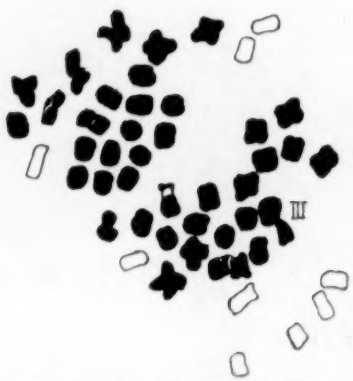
This clone is an F_1 hybrid between Vellai (*S. officinarum*, n=40) and *S. spontaneum* Coimb. (n=32). According to Dutt and Rao,[13] 56 bivalents were formed in metaphase I of pollen mother cells. Bremer[11] counted 109-112 chromosomes in root tip cells of this clone. The author found three P.M.C.'s with 56_{II}, three P.M.C.'s with $55_{II}+2_I$, and one P.M.C. with $53_{II}+6_I$. In some of these P.M.C.'s, however, a few bivalents appeared to be loosely connected and formed quadrivalents. In diakinesis one or two quadrivalents were observed in each of the P.M.C.'s observed. This hybrid has 112 chromosomes in its somatic cells, 40 chromosomes being in excess of the expected ($2n$=72).

(4) H 109 (n=40) × *S. spontaneum* Coimb. (n=32)?—H 44-394

This clone was listed at the Experiment Station, H.S.P.A., as a hybrid obtained from the cross H 109 × *Narenga porphyrocoma*. The chromosome number (probably $2n$=112, Dr. Price, unpublished), however, suggests that this parentage is very questionable. Upon rechecking the field notes recorded at the time of crossing it was concluded that this clone is probably a hybrid between H 109 and *S. spontaneum* Coimb. (n=32).

This conclusion is also supported by the author's cytological study on the meiosis of P.M.C.'s. Four P.M.C.'s showed $55_{II}+2_I$, two showed $54_{II}+4_I$, and one showed 56_{II} in metaphase I. The number of univalents varied from zero to four. One to four quadrivalents per cell were usually found, but sometimes none was observed. The behavior of chromosomes in succeeding stages of meiosis in this hybrid was similar to that described above in other hybrids.

In short, of the four *S. officinarum* × *spontaneum* Coimb. hybrids, one had 31 chromosomes and three had 40 chromosomes in excess of the expected number (40+32=72=$2n$). In the former it seems most probable that the



CHROMOSOMES IN METAPHASE I OF LOETHERS

Figure 23

The fundamental configuration seemed to be $40_{II} + 10_{I}$ in this variety, but some modifications were induced by the formation of one to five trivalents. $1_{III} + 39_{II} + 9_{I} = 90$.

chromosome doubling might have taken place in chromosomes from the pollen parent, although one chromosome was missing; that is, 40+(32+31)=103. In the latter three hybrids, the chromosomes from the female parent have been duplicated; that is, (40+40) +32=112. In these three plants 40_{II} are probably formed by the double number of *officinarum* chromosomes. If the chromosomes of *S. spontaneum* Coimb. (*n*=32) formed 16_{II}, through autosyndesis, 56_{II} would be found in metaphase I of these hybrids. However, it is probable that one or two pairs of *spontaneum* chromosomes would fail to pair in some cases.

In the hybrid with 2*n*=103 [40+(32+31)], $31_{II}+1_{I}$ are probably formed by the chromosomes from *S. spontaneum* Coimb. and 20_{II} are probably due to autosyndesis among *officinarum* chromosomes, where a few bivalents sometimes fail to pair.

(5) *S. robustum* (*n*=40) × *spontaneum* Coimb. (*n*=32)—H 36-1102

Only a little of the *S. robustum* × *spontaneum* Coimb. material was available; therefore, observations (Table I) were made on a limited number of P.M.C.'s. In first metaphase $35_{II}+2_{I}$ and in some cases $34_{II}+4_{I}$ were found. The total number of chromosomes is 72, which is the sum of the haploid number of chromosomes from both parents. In ana-telophase I, zero to six lagging univalents were observed. The frequency of their occurrence seems to be higher than in any other of the hybrids shown above. It was noted that halves of some of the longitudinally divided laggards did not disjoin, but were attached to each other at one end and were stretched between the two poles. Unequal separation or breakage of chromosomes was occasionally found in telophase I.

In this hybrid it is most probable that the formation of bivalents is due to the autosyndesis of chromosomes from both parents though one or a few pairs of chromosomes often fail to form bivalents.

2. *Some other interspecific hybrids*

In the course of the cytological study, the author observed some interspecific hybrids which had excessive aneuploid numbers of chromosomes. The meiotic behavior of chromosomes in these hybrids will be briefly described below (Table I).

(1) *S. spontaneum* (Burma, *n*=48) × 247 B (*S. officinarum*, *n*=40)—H 51-9001

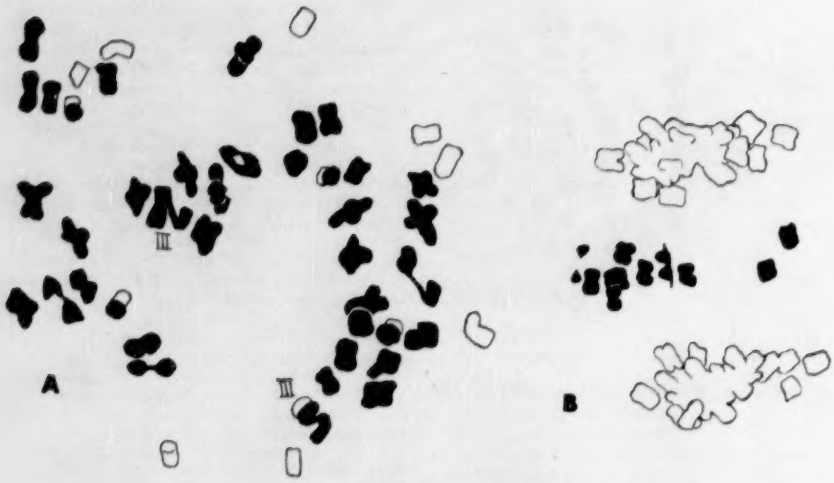
According to Bremer[1,2] and Dr. Price (unpublished) the clone, 247 B, has 40 haploid

TABLE I. Chromosome configurations in meiosis of *Saccharum* varieties and hybrids

Line No.	Species or parentage	2*n*	Chromosome configuration I*	II*	Remarks	Unexpected increase in chromosome number
H 51-9457	*S. officinarum* (*n*=40) (H 109) × *spontaneum* Coimb. (n=32)	103	1	50	$0–1_{III}$	+31
H 51-9035	*S. officinarum* (n=40) (D 908) × *spontaneum* Coimb. (n=32)	112	2	55	56_{II} in rare case	+40
Co. 205	*S. officinarum* (*n*=40) (Vellai) × *spontaneum* Coimb. (n=32)	112	0–2	56–55	$0–1_{III}$? $0–4_{IV}$, (5_{IV})	+40
H 44-394	*S. officinarum* (*n*=40) (H 109) × *spontaneum* Coimb. (*n*?=32)†	112	2–4	55–54	56_{II} in rare case, $0–4_{IV}$	+40
H 36-1102	*S. robustum* (*n* = 40) × *spontaneum* Coimb. (*n*=32)	72	2–4	35–34		0
H 51-9001	*S. spont.* Burma (*n*=48) × *officinarum* (247 B)	101	9	46	$0–3_{III}$, (4_{III})	+13
H 53-9084	SW 111(?) × *spont.* Coimb. (*n*=32)	85	3	41	$0–3_{III}$	+13?
H 53-9081	SW 111(?) × *spont.* Coimb. (*n*=32)	83	3	40	$0–3_{III}$, 1_{IV}?	+11?
H 33-8072	*S. officinarum* (*n* = 40) × *robustum* (*n*=prob. 40)	93	1–5	46–44	$0–1_{III}$, $0–1_{IV}$	+13
100 POJ	100 POJ	88	10	39	$2_{III}–5_{III}$, (6_{III})	+ 8
Loethers	Loethers	90	10	40	$0–3_{III}$, (5_{III}), $0–1_{IV}$	+10
H 109	*S. officinarum*	80	0	40	1_{IV}?	0

*The usual number of bivalents (II) and univalents (I) is shown.

†Probably *S. spontaneum* Coimbatore



MEIOTIC PAIRING IN 100 P.O.J.

Figure 24

A—Chromosomes in metaphase I of 100 P.O.J. $2_{III} + 37_{II} + 8_I = 88$. *B*—Anaphase I of the same plant, showing a pair of small lagging chromosomes (fragments) separated longitudinally.

chromosomes. The expected number of chromosomes in this hybrid is 48+40=88 (*2n*). The actual number, however, was found to be *2n*=101, 13 chromosomes being in excess. The meiotic configuration is $46_{II}+9_I$. One to four univalents seemed sometimes to be loosely associated with bivalents. Most of the univalents divided longitudinally in ana-telophase I and moved to opposite poles. In telophase II chromosome breakage was sometimes observed.

(2) SW 111 (?) × *S. spontaneum* Coimb. (*n*=32)

SW 111 is a hybrid between two varieties of *S. officinarum*, Black cheribon (*n*=40), and Batjan (*n*=40), and its chromosome number is *2n*=80, *n*=40 (Bremer[3,4]). However, the female plant used in this cross was designated as SW 111, but its chromosome number is *2n*=106-112 (Dr. Price, unpublished). This clone is probably different from the original one studied by Bremer.

Two sister plants from the cross SW 111 (?) × *S. spontaneum* Coimb. were studied. One hybrid, H 53-9081, showed $40_{II}+3_I$ and in extreme cases $3_{III}+37_{II}$, 83 being the somatic number. In the other hybrid, H 53-9084, the chromosomes in anaphase I of one P.M.C. were clearly counted to be 43+42=85 (*2n*), and in another cell, 44+½ (longitudinal half) +½+40=85 (*2n*). The chromosome configuration was usually $41_{II}+3_I$, or $3_{III}+38_{II}$ in rare cases in diakinesis or metaphase I.

We have no knowledge of the meiotic pairing of chromosomes in the female parent. Probably more than 40 bivalents are formed, in which case the female parent would have contributed more than 40 chromosomes (probably 40+11 or 13) to the hybrids.

(3) Badila (*n*=40) × *S. robustum* (Probably *n*=40)—H 33-8072

The fixed material from this hybrid was kindly supplied by Dr. Price. According to him the male parent may have *n*=40, because all of its sister plants have 40 haploid chromosomes. This hybrid showed $46_{II}+1_I$ to $44_{II}+5_I$ in metaphase I. The formation of trivalents was not clear, but a few quadrivalents were found in rare cases. The somatic number can be assumed to be 93, 13 chromosomes being in excess.

Other instances are already recorded in which an aneuploid increase in the chromosomes occurred in an artificial crossing of *Saccharum;* however, there is no exact knowledge on the mechanism by which this aneuploid comes about.

3. *Some sugarcane clones*

(1) Loethers

According to Bremer,[7] Loethers is not a pure descendant from *S. officinarum*. In his study of meiosis Bremer[3,4] failed to observe the chromosome configuration. He suggested from observations on anaphase I that the chromosome number is *2n*=99. In the present

investigation, the author made careful observations on chromosome pairing in diakinesis and metaphase I of Loethers. Occasionally it is difficult to determine whether a certain configuration is a bivalent or a trivalent. Accordingly, the somatic number varied from 89 to 91; that is, two P.M.C.'s showed $2n$=89-90, five showed $2n$=90, one showed $2n$=90-91. The fundamental configuration seemed to be $40_{II}+10_{I}$ and some modifications were induced by the formation of one to five trivalents (Figure 23).

(2) 100 P.O.J.

This clone is probably a progeny from Bandjermasin hitam (n=40; Bremer[1,2])×Loethers (n=99/2; Bremer[4,10]). This is a grandmother of the famous commercial canes, 2728 P.O.J., 2883 P.O.J., and 2725 P.O.J. From his observations on meiotic chromosome pairing (ca. $38_{II}+13_{I}$, etc.) and on root tip cells ($2n$=88-90), Bremer[10] concluded that the most probable number is $2n$=89.

The author's observations suggest that the basic pairing is $39_{II}+10_{I}$=88. However, this pairing is modified by univalents which conjugate with bivalents to form trivalents. The number of trivalents varied from two to five, rarely six (Figure 24*A*). One univalent is very small and resembles a fragment (Figure 24*B*). The number, $2n$=88, observed in metaphase I, agrees with a count made in anaphase I; *i.e.*, 43+5+40=88. It is a very interesting fact that no more than 39 chromosome associations were found in 100 P.O.J. One might suppose that at least 79 *officinarum* chromosomes may be present, forming $39_{II}+1_{I}$.

TABLE II. A review of the basic number of polyploidy in *Saccharum*

Author	Year	Basic number (x)
Bremer[5, 6]	1924	10, (5)
Dutt and Rao[12]	1933	8
Janaki-Ammal[15]	1936	5 or 10 and 6 or 12
Janaki-Ammal[16]	1939	10 and 6
Darlington and Janaki-Ammal[12]	1945	10 and 12
Parthasarathy[20]	1948	5
Raghavan[22]	1951	5
Nishiyama (present paper)		8 and 10

(3) H 109

This cane variety was obtained in the progeny of Lahaina, which belongs to *S. officinarum*. Its chromosome number was already reported to be n=40, $2n$=80 (Weller and Mangelsdorf[38]). The author's observation agrees very well with those results; that is, 40_{II} were found, and there were no irregularities in chromosome pairing, except the rare occurrence of a few quadrivalents.

Discussion

The remarkable cytology of the genus *Saccharum* was first described by Bremer,[1,2] whose work has great theoretical and applied importance. Owing to high chromosome numbers, however, cytogenetic studies were made with much difficulty. The difficulty is illustrated by the fact that the basic number of this polyploid group is still undecided (Table II). There are four principal methods by which the basic number in polyploid groups can be determined.

(1) A simple arithmetical consideration of polyploid series: The largest common divisor of the number in a polyploid series is usually adopted as the basic number.

(2) A basic number is indicated by chromosome homologies exhibited in meiosis of F_1 hybrids, the method of genome analysis proposed by Kihara.[20]

(3) A basic number is indicated by secondary polyploidy which is suggested by the secondary association of chromosomes.

(4) The karyotype analysis, or "basikaryotype" analysis (Sinoto and Sato[36]) can be applied to the determination of basic number.

One may arrive at the basic chromosome number by one of these methods, but such a basic number must be verified by other information.

In *Saccharum* the basic number has already been discussed from the standpoint of the first three methods given above. From a consideration of chromosome numbers, Bremer[5,6,8] proposed that 10 is the basic number

TABLE III. Chromosome configurations of hybrids in *Saccharum* and its relatives

Hybrids	Chromosome configurations*	$2n$	Unexpected increase in chromosome number	Authors
S. off. (Vellai) × *spont.* Coimb.	56_{II}	(40+40)+32=112	+40	Dutt and Rao[12]
S. off. (H 109 etc.) × *spont.* Coimb.	$(54–56)_{II}+(4–0)_{I}$	(40+40)+32=112	+40	The author
S. off. (H 109) × *spont.* Coimb.	$50_{II}+3_{I}$	40+(32+11)=103	+11	The author
S. officinarum × *spont.* Glagah Java	$(62–66)_{II}+(12–4)_{I}$	(40+40)+56=136	+40	Bremer[1,2,7]
S. officinarum × *spont.* Glagah Java	$60_{II}+16_{I}$	(40+40)+56=136	+40	Nishiyama *et al.*[37]
S. officinarum × *spont.* Glagah Java	$66_{II}+6_{I}$	(40+40)+58=138	+40(+2)	Li *et al.*[21]
2725 POJ × *spont.* Ponape	$(71–72)_{II}+(4–2)_{I}$	106+40=146	+51	Nishiyama *et al.*[37]
S. robustum × *spont.* Coimb.	$(34–35)_{II}+(4–2)_{I}$	40+32=72	0	The author
S. spont. Karenko × *spont.* Glagah Java	$48_{II}+8_{I}$	48+56=104	0	Nishiyama *et al.*[37]
S. off. (Vellai) × *Narenga porphyrocoma*	$(25–26)_{II}+(5–3)_{I}$	40+15=55	0	Janaki-Ammal[18]
S. spont. (Glagah Java) × *Erianthus ravennae*	$(28–29)_{II}+(10–8)_{I}$	56+10=66	0	Janaki-Ammal[17]

*The usual or highest number of bivalents is shown. The number of multivalents is omitted.

of *Saccharum*. According to Bremer,[5] five is theoretically possible, because the basic number in Andropogoneae and Maydeae is five. Other chromosome numbers which differ from this exact multiple series, *e.g.*, $n=56$ of *S. spontaneum*, were suggested to be aneuploid (chromosome aberrations).

Dutt and Rao[13] counted chromosome numbers in *S. spontaneum* of India and found that the polyploidy shows multiples of eight.

From her study on the secondary association of chromosomes, Janaki-Ammal[15,16] pointed out that *Saccharum* has two different kinds of basic numbers as shown in Table II. Later, Darlington and Janaki-Ammal[12] modified her earlier opinions and suggested the basic number to be $x=10$ and 12.

Parthasarathy[28] tried to analyze *officinarum* chromosomes by the backcross method in which a *S. officinarum* × *Sclerostachya fusca* F_1 was backcrossed to *Sclerostachya* generation after generation. He reported that 10 *officinarum* chromosomes, usually assumed to be one chromosome set, were further divided into two sets, each possessing five chromosomes. One of the two sets was said to be homologous with one of the three genomes of *Sclerostachya fusca* ($n=15$), which shows a basic number of five.

Raghavan[32] made a similar study and came to the conclusion that two sets of five basic chromosomes of *S. officinarum* were homologous with two sets of *Narenga* ($n=15$), which possesses three sets of five chromosomes. According to Raghavan,[32] 15 *Narenga* chromosomes are homologous to those of *Sclerostachya*.

Below is described the author's attempt to analyze the genomes in *Saccharum* by studying meiotic chromosome homologies in hybrids. Recently numerous reports on the cytology of sugarcane have appeared. In most cases, however, one of the parents was a complex hybrid obtained through one or more generations of artificial crossing or "nobilization." In these crosses, genome analysis is often fraught with considerable complexity. Table III shows the results of meiotic studies in cases in which the hybrids were relatively simple.

A. *S. officinarum* ($n=40$) × *spontaneum* Coimb. ($n=32$)

There are two kinds of F_1 hybrids from this cross, those hybrids having $(40+40)+32=112$ chromosomes ($2n$) and the hybrid with $40+(32+31)=103$ chromosomes ($2n$). Generally speaking, the former showed 56_{II}, which might be due to the sum of 40_{II} (synapsis of *officinarum* chromosomes) plus 16_{II} (autosyndesis of *spontaneum* chromosomes). In the hybrid with $2n=103$, there are found $50_{II}+3_I$ which may be due to 20_{II} (autosyndesis of *officinarum* chromosomes)$+31_{II}$ (synapsis of *spontaneum* chromosomes)$+1_I$ (*spontaneum* chromosomes), but often one bivalent fails of pairing. The occurrence of autosyndesis in *officinarum* chromosomes can be clearly seen in the intergeneric hybrid, *S. officinarum* ($n=40$)×*Narenga porphyrocoma* ($n=15$) (Janaki-Ammal[18]). From these results we can suppose that the 40 haploid chromosomes of *S. officinarum* are a duplication of 20 chromosomes. In the same way, the 32 *spontaneum* chromosomes are considered to be a duplication of 16 chromosomes. We shall further consider the mode of chromosome pairing from the point of view of the basic number. The 40 chromosomes (n) of *S. officinarum* might be considered as multiples of either eight or 10. Assuming that no multivalents would occur, autosyndetic pairing among 40 *officinarum* chromosomes would depend upon whether the basic number was eight or 10 as follows:

$n=40$	$8+8+8+8+8$	$10+10+10+10$
autosyndesis	$16_{II}+8_I$	20_{II}

In *S. spontaneum* Coimb. ($n=32$), three basic numbers $x=8$, 10, and 12 are taken under consideration (two is an aneuploid number).

$n=32$	$8+8+8+8$	$10+10+10+2$	$10+10+12$
auto-	16_{II}	$10_{II}+12_I$	$10_{II}+12_I$
syndesis		or	
		$12_{II}+8_I$	

From these theoretical considerations of chromosome pairing, it seems that the basic numbers 10 in *S. officinarum* and eight in *S. spontaneum* Coimb. show the best agreement with observed results.

B. *S. officinarum* ($n=40$) × *spontaneum* Glagah Java ($n=56$)

Unfortunately, the author has had no opportunity to observe meiosis in this type of hybrid. Since Bremer's remarkable studies on the hybrid, Kassoer, natural hybrids or artificial hybrids from similar crosses have been studied by many workers; all of them reached the same conclusion. In this cross, doubling of *officinarum* chromosomes usually occurred as follows: $(40+40)+56=136$ ($2n$). As suggested by Bremer,[7] 40_{II} (*officinarum* chromosomes)$+28_{II}$ (autosyndesis of *spontaneum* chromosomes)$=68_{II}$ are possible in principle. Actually, wide variations of the number of univalents are usually found in metaphase I (Table III). It appears that some *spontaneum* chromosomes often fail to pair. In the hybrid between *S. spontaneum* ($n=56$) and *Erianthus ravennae* ($n=10+1$fr.), however, $28_{II}+10_I$ (probably autosyndesis of *S. spontaneum*) were almost always formed (Janaki-Ammal[17]). From these results one can not determine whether it is *officinarum* or *spontaneum* chromosomes that fail to pair in Kassoer or in similar hybrids. It can be said, however, that

more homology actually exists among the chromosomes of *S. officinarum* × *spontaneum* hybrids than is suggested by their meiotic pairing.

Among several combinations of hypothetical basic numbers (8, 10, or 12) only two can be proposed to explain the 56 chromosomes (n) of Glagah Java as follows:

n=56 8+8+10+10+10+10
$2_{8_{II}}$ (autosyndesis)
n=56 8+8+ 8+ 8+12+12
$2_{8_{II}}$ (autosyndesis)

C. 2725 P.O.J. ($2n$=107) × *S. spontaneum* Ponape (n=40)

This hybrid has 146 chromosomes ($2n$), 106 of which were probably derived from doubling of 53 chromosomes (reduced number) of 2725 P.O.J. and the remaining 40 from *S. spontaneum*. The meiotic pairing, $(71\text{-}72)_{II}+(4\text{-}2)_I$, of chromosomes in this hybrid also strongly suggests the possibility of autosyndesis among 40 Ponape chromosomes. Thus, just as in *S. officinarum*, the 40-Ponape chromosomes can be assumed to have four of 10 basic chromosomes.

Bremer[7] attempted an observation on meiosis in the hybrid ($2n$=120) between *S. officinarum* (Ardjoeno, n=40) and *S. spontaneum* (Glagah Tabongo n=40) but could not ascertain the mode of chromosome pairing.

D. *S. robustum* (n=40)×*spontaneum* Coimb. (n=32)

This hybrid showed no doubling of chromosomes. The somatic number of chromosomes is 40+32=72, and meiotic pairing is usually $34_{II}+4_I$ or $35_{II}+2_I$. This fact is suggestive of the occurrence of autosyndesis in chromosomes from both parents. In *S. robustum* the basic number, 10, is the most probable just as in *S. officinarum*.

E. *S. spontaneum* Karenko (n=48) × *spontaneum* Glagah Java (n=56)

This hybrid has the sum of haploid chromosomes of both parents and generally shows $48_{II}+8_I$ in metaphase I. If one assumed that both Karenko and Glagah Java contained only multiples of eight, this hybrid would always show $48_{II}+8_I$ whether by auto- or allosyndesis. Although this chromosome configuration agrees with cytological observations, the above constitution would not allow the 56 chromosomes of Glagah to form 28_{II} by autosyndesis. This point has been mentioned above.

Now, other possibilities follow:

$$n=48 \begin{cases} 8+10+10+10+10 \\ 8+\ 8+\ 8+12+12 \end{cases}$$

$$n=56 \begin{cases} 8+8+10+10+10+10 \\ 8+8+\ 8+\ 8+12+12 \end{cases}$$

From these considerations the most probable basic numbers seem to be eight and 10. The probable genome constitutions of some *Saccharum* species are shown in Table IV (see Nishiyama *et al*[21]). The basic numbers, eight and 10, are likely to be applicable to most other cytological observations in *Saccharum*.

From the cytological observations presented here, it can not be said that the 8-chromosome sets of Karenko and of Glagah Java are homologous with each other. It is not yet certain whether the four 8-chromosome sets in Coimbatore are duplications of two different sets or not. The same problem arises in connection with the 10-chromosome set. Li and Leung[22] studied this problem in the three-way hybrids but reached no final conclusion. It would be interesting to know the homology between the 10-chromosome sets of *S. officinarum* with those of other species. This would be of special importance in breeding programs. It would also be very interesting to know what relationship exists between 8- and 10-chromosome sets. Perhaps in further studies on genomic evolution in *Saccharum* 10-chromosome sets should be divided into two pro-genomes. The hypotheses of a 5-chromosome basic number proposed by Parthasarathy[28] and by Raghavan,[32] although suggestive, require further studies for verification.

TABLE IV. Genome constitution of some *Saccharum* species

Species	n	Genomes (x=8 and 10)
S. spontaneum Coimbatore	32	8 8 8 8
S. spontaneum Karenko	48	8 10 10 10 10
S. spontaneum Glagah, Java	56	8 8 10 10 10 10
S. spontaneum Ponape	40	10 10 10 10
S. officinarum	40	10 10 10 10
S. robustum	40	10 10 10 10

Summary

Cytological observations, especially meiotic pairing of chromosomes, were made on three clones and nine interspecific hybrids in *Saccharum*.

The chromosome numbers of 100 P.O.J. and Loethers did not agree with those previously reported.

In *S. officinarum* (n=40) × *spontaneum* Coimbatore (n=32), the doubling of the chromosomes from the female parent was always observed with exception that one showed duplication of the chromosomes of the male parent.

In one hybrid, *S. robustum* (n=40)×*spontaneum* Coimbatore (n=32), there was found no increase of chromosomes.

Some hybrids showed an increase of an aneuploid number of chromosomes, but the origin of the aneuploid number of chromosomes is not clear.

The analysis of genomes in *Saccharum* has been discussed. Based on homologies of chromosomes in hybrids, it can be said that in *Saccharum* there are two basic numbers, eight and 10. Polyploidy in *Saccharum* con-

sists of multiples and combinations of these two basic numbers.

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NO CLEFT PALATES IN OFFSPRING OF PREGNANT MICE GIVEN CORTISONE AFTER FETAL PALATE CLOSURE

Daphne G. Trasler, Karin H. Clark and F. C. Fraser*

PREVIOUS work from this laboratory has shown that cleft palates can be produced in the offspring of pregnant female mice treated with cortisone acetate before the time of normal embryonic palate closure. A few cleft palates were found in the offspring of females that were said to have been treated after this time.[1] These will be referred to as post-closure clefts. Since the production of palatine clefts by reopening of already closed palates would have important teratological implications, further data have been gathered to find out if post-closure clefts do, in fact, occur.

Methods and Results

As in the previous work[2] mice of the inbred strain A/Jax, which is highly susceptible to the teratogenic effects of cortisone, and the relatively resistant inbred C57BL/Fr,[3] were used. Post-closure clefts had been reported as occurring in both these strains. Also treated were F_1 females from the reciprocal crosses between the A/Jax and DBA/1 strains, backcrossed to A/Jax males, and CAF_1 hybrid females crossed to their brothers. (CAF_1 hybrids are the F_1 of the cross BALB/c × A/Jax.) The offspring of these crosses have high frequencies of cleft palates when cortisone is administered to the pregnant mothers some days prior to embryonic palate closure.[2]

The mice were kept under the same conditions as in the previous experiment. All experimental females were examined daily for vaginal plugs, the day the plug was observed being called day 0 of gestation. Embryological studies have shown that the palate is normally closed by the afternoon of day 15, in the A/Jax and C57BL animals.[4] Treatments were therefore started on the afternoon of day 15, or on day 16 or 17. The dose range, 2.5, 5, or 10 mg. of cortisone acetate a day for one or more days was similar to that used in the previous work.

The controls consisted of young born to untreated mothers of the same strains.

The 108 treated females gave birth to 525 young, all of which had normal palates (Table I). Among the controls

*Department of Genetics, McGill University, Montreal, Canada. We wish to thank Dr. H. Kalter for providing the greater part of the data for the A/Jax with DBA/1 crosses, and Dr. J. H. Laurie of Merck & Company for kindly supplying us with cortisone. The financial assistance of the Kate E. Taylor Fund of the Banting Research Foundation and of the National Research Council of Canada is gratefully acknowledged.

(Table II) seven out of 2501 young had cleft palates without cleft lips, a spontaneous frequency of 0.28±0.11 percent. (The relatively few newborns with cleft lips, with or without cleft palates, were excluded from the data.)

Summary and Conclusions

The present experiment has failed to show that cortisone acetate can produce cleft palates when given to pregnant females after the normal time of embryonic palate closure. From the control data it is seen that occasionally spontaneous cleft palates occur. The spontaneous rate, however, is too low to account for the three percent frequency of clefts previously reported to be produced by cortisone treatment after palate closure. Since the timing of gestational stage in the previous work was not by vaginal plug, but by palpation, or by counting back from day of birth (assuming a uniform gestation period), it is probable that some, if not all, of the previously reported cases of post-closure clefts were actually treated before the palate closed.

It appears highly unlikely that cortisone acetate can induce cleft palates in the offspring of pregnant female mice treated after the normal time of embryonic palate closure.

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TABLE II. Spontaneous incidence of cleft palate (C.P.) without cleft lip in the control animals

Mating ♀	Mating ♂	Number of ♀♀	Total* young	Number with C.P.	% C.P.
A/Jax	A/Jax	78	433	1	0.23
(A/Jax × DBA)	A/Jax	151	966	3	0.31
(DBA × A/Jax)	A/Jax				
CAF_1	CAF_1	4	26	0	0.00
C57BL	C57BL	174	1076	3	0.28
	TOTAL	407	2501	7	0.28

*All young having cleft lip have been excluded.

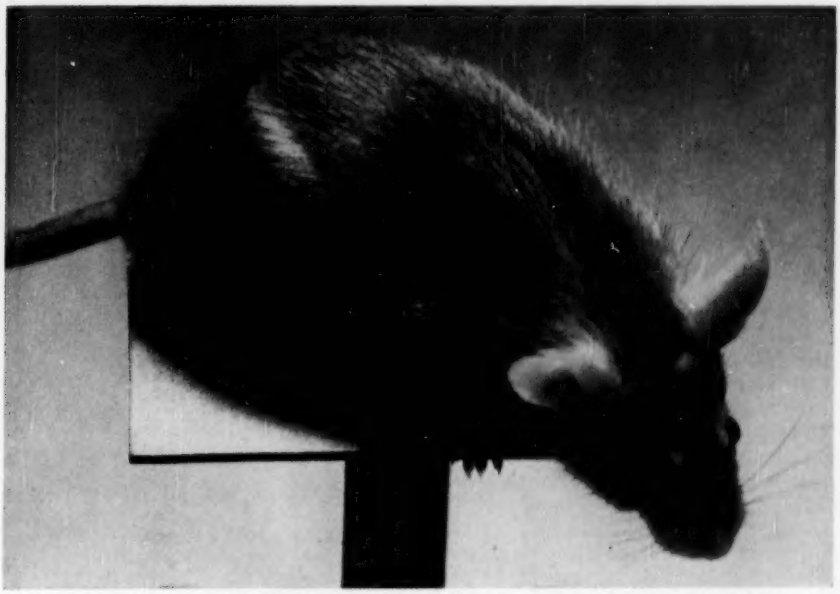
TABLE I. Frequency of cleft palate (C.P.) in the offspring of pregnant mice treated with cortisone after the time of embryonic palate closure

Mating ♀	Mating ♂	Gestation day treated	Daily dose in mg.	No. of doses	No. ♀♀	Total* young	No. with C.P.
A/Jax	A/Jax	15	2.5	3	1	6	0
			5.0	1	1	3	0
(A/Jax × DBA)	A/Jax	15	2.5	3	1	7	0
(A/Jax × DBA)	A/Jax	15	2.5	4	4	20	0
(DBA × A/Jax)	A/Jax						
A/Jax	A/Jax	16	2.5	2	1	8	0
			2.5	3	4	21	0
			2.5	4	6	29	0
(DBA × A/Jax)	A/Jax	16	2.5	1	1	3	0
(A/Jax × DBA)	A/Jax	16	2.5	2	8	40	0
(DBA × A/Jax)	A/Jax						
(A/Jax × DBA)	A/Jax	16	2.5	3	1	8	0
CAF_1	CAF_1	16	2.5	3	1	10	0
C57BL	C57BL	16	2.5	3	1	7	0
			2.5	4	2	7	0
A/Jax	A/Jax	17	2.5	2	3	15	0
			2.5	2	1	10	0
			5.0	1	4	20	0
(A/Jax × DBA)	A/Jax	17	2.5	2	2	15	0
			10.0	1	2	8	0
C57BL	C57BL	17	2.5	2	5	29	0
			2.5	3	5	28	0
			2.5	4	2	7	0
			2.5	5	1	3	0
			5.0	2	27	125	0
			10.0	1	24	96	0
	TOTAL				108	525	0

*All young having cleft lip have been excluded.

HEREDITARY HAIR LOSS IN THE TUFTED MUTANT OF THE HOUSE MOUSE

MARY F. LYON*



TUFTED—A NEW CHROMOSOME MARKER IN LINKAGE GROUP IX
Figure 25
Adult tufted male showing three waves of hair loss—one around the eyes, one on the forehead and one on the haunches.

THE mutant tufted, symbol *tf*, of *Mus musculus* is a new recessive in linkage group IX characterized by repeated loss and regrowth of hair (Figure 25).

The first mutant animal was found among the descendants of a male given 700r of X-rays to the testis in an experiment intended to induce translocations.[2] However, the pedigree indicates that the mutation was almost certainly spontaneous and not induced by the radiation. The irradiated male was outcrossed to a female of a multiple recessive stock and his sons were then backcrossed to the same stock. The first tufted animal appeared in the offspring of a backcross mating. As *tf* was later proved to be recessive, it must have been carried by the female used for the backcross; the simplest hypothesis, therefore, is that *tf* arose spontaneously in the recessive stock at some time before the outcross to the irradiated male, and was transmitted by both the females used.

Description

Tufted is first detectable at about four weeks, when tufted mice begin to lose the baby coat. A wave of hair loss begins ventrally under the jaw and spreads

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posteriorly, followed a few days later by a similar dorsal wave beginning on the face; the sinus hairs are not shed. A wave of growth of the second coat follows closely behind the waves of loss. This second coat is completely normal in texture and structure of the individual hairs (as also are the baby coat and all later coats) and for a short time after it has grown the mouse may be indistinguishable from normal. Then the second coat is lost in an antero-posterior wave and the third coat grows in. This cycle of hair growth and loss is repeated throughout life.

Measurements of the times of waves were made on 10 animals up to three months of age. A wave of loss and regrowth passed from the ventral jaw to dorsal haunches region in seven days for the first wave, 18 days for the second and 28 days for the third wave. The longer duration of the later waves seemed due to a longer interval between successive replacements of hairs in the dorsal and posterior parts of the body. Between one and three months of age the time between successive waves was, for example, 12-16 days for the throat, 18-20 days for the face and 22-24 days for the haunches. Thus, later waves begin anteriorly before earlier ones have reached the haunches and two or more waves may be passing over the animal at the same time (Figure 25). The number of waves present does not increase indefinitely however, so it seems probable that after three months of age the times between waves in various parts of the body change once more. During pregnancy and lactation neither loss nor growth of hair occurs, and the cycle begins again at the end of lactation.

No histological studies have been made, and it is not known whether the hairs break off or fall out of the follicles. However, the hairs which are shed appear completely normal when mounted in Canada balsam and examined microscopically.

TABLE I. Single factor segregation of tufted

Mating type	Offspring +	*tf*
tftf × ++	16	—
+*tf* × +*tf*	10	3
+*tf* × *tftf*	85	88
tftf × *tftf*	—	48

Genetics

The single factor segregations of tufted given in Table I show that it is inherited as a simple autosomal recessive; there is no evidence of reduced viability or incomplete penetrance.

Linkage tests (Table II) show that tufted is closely linked to brachyury, *T*, in linkage group IX. In coupling backcrosses the recombination of *tf* with *T* was 8.3±1.5 percent. When the heterozygous parents were divided according to sex the recombination was found to be 7.7±1.8 percent in females and 9.2±2.4 percent in males; the two values are not significantly different. The previously known genes in linkage group IX are *H-2—Fu—T*.

The recombination between *H-2* and *T* is 15 percent in female heterozygotes and 8 percent in males.[8,1] Tufted may therefore lie either close to *H-2* or beyond *T*. No tests of its position have been made.

TABLE II. Linkage tests with tufted

Mutant *m—M*	Mating type*	Progeny ++	*m*+	+*tf*	*mtf*	Total	R.F.	χ^2	S.E.
a	BC	23	27	29	24	103	54.4	0.786	4.9
b	BC	36	46	41	40	163	53.4	0.742	3.9
c^{ch}	BC	41	40	40	37	158	50.6	0.025	4.0
d	IC	26	7	21	11	65	37.0	1.473	10.7
		M+	++	*Mtf*	+*tf*				
Ca	BC	30	20	30	23	103	48.5	0.087	4.9
Ra	BC	5	11	9	8	33	60.6	1.485	8.7
Re	BC	12	23	20	15	70	61.4	3.657	6.0
T	BC♀	95	7	10	108	220			
	♂	70	4	9	58	141	8.3	251.0	1.5
W^v	BC	16	9	19	9	53	52.8	0.170	6.9
Mf^{wh}	BC	28	22	31	22	103	51.5	0.087	4.9
	BR	3	6	4	6	19	47.4	0.053	11.5

*B = backcross for both mutants; I = intercross for *d*, backcross for *tf*; C = coupling; R = repulsion.

Comparison with Other Mutants

Other genes causing hair loss in the mouse include alopecia, *Al*, naked, *N*, hairless, *hr*, rhino, hr^{rh}, and furless, *fs*. Naked lies in linkage group VI close to caracul; hairless in linkage group III close to spotting; and alopecia gives indications of linkage with group VII.[4] Hence, from its linkage relations, tufted is not allelic with any of these mutants. It bears a phenotypic resemblance to naked except that tufted animals usually have more hair present at any time than naked animals. The hairs of naked mice have "weak spots" due to poor keratinization,[3,7] whereas those of tufted mice are normal. Furless is phenotypically very different from tufted; the hairs and vibrissae are shortened, the first coat is shed during the third week of life, and after shedding the second coat the mouse remains furless throughout life.[9] It thus seems improbable that tufted is allelic with furless, but no tests have been made.

Discussion

Tufted is of interest in two ways. First, since it is phenotypically and genetically distinct from previously described genes, it represents a new type of hereditary hair loss in the mouse and is potentially capable of giving new information about normal hair growth. Secondly, as a new chromosome marker in linkage group IX it may be useful in studying the problems of the *t*-series of alleles, some at least of which have been shown to reduce crossing-over in their neighborhood.[5,6]

Summary

Tufted, *tf*, is a recessive mutant which causes repeated waves of hair loss and regrowth, which pass antero-posteriorly along the body. Individual hairs are normal. Viability and penetrance are good. Tufted lies in linkage group IX, and shows 8.3 percent recombination with *T*.

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LEAF MARKING ALLELES IN WHITE CLOVER

Uniform Nomenclature

J. L. Brewbaker and H. L. Carnahan*

THE genetic control of white clovers' *V*-leaf markings has been the subject of recent papers by Carnahan *et al.*[4] and Brewbaker[2]. Atwood and co-workers[1] at the U. S. Regional Pasture Research Laboratory first reported studies of white clovers' leaf markings and designated the controlling locus *V* as in red clover. Brown† and Carnahan *et al.*[4] described eight *V* alleles among Pasture Laboratory material, to which Brewbaker and Anderson[3] and Brewbaker[2] later added two allelomorphs and Pandey (cited by Brewbaker[2]) a third.

Certain inconsistencies in the usage of symbols for the 11 *V* alleles appeared in these publications, and the authors[2,4] in order to

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†Paper presented by K. G. Brown at the meeting of the American Society of Agronomy in February, 1946.

avoid confusion are adopting the symbols listed below:

Adopted Allelic Symbol	Symbols formerly used by: † and 4	2 and 3	Phenotypic Designation
v	v	v	No marking
V^h	V^h	V^H	Full V—high
V^i	V^i	—	Full V—intermediate
V^l	V^l	V	Full V—low
V^b	V^b	V^{Bh}	Broken V
V^{bd}	—	V^B	Broken V—dominant
V^p	V^p	V^P	V—point
V^f	V^{wi}, V^{fi}	V^p, V^{fi}	Filled V
V^{bc}	—	V^L	Basal V
V^{by}	V^{by}	—	Broken V with yellow tip
V^{fg}	—	—	Filled V with green base

The symbol v has been used by all authors to describe the no marking phenotype of the recessive homozygote vv. The symbols V^h, V^i, and V^l were applied by Brown† with supporting data by Carnahan *et al.*[4] to three types of full V-markings. Further tests are required to distinguish with certainty Brewbaker's full V type (his allele V) from the V^i (full V—intermediate) and V^l (full V—low) of Brown, although available data favor its identity with V^i. Both studies suggest allele V^i to be the most common of the alleles producing a marking, and of nearly equal frequency to v.

Two broken V markings were discerned in the material of Brewbaker[2], while Brown† described but a single broken V allele which was designated V^b. The data published by Carnahan *et al.*[4] made it possible to identify Brewbaker's "broken V, high" (V^{Bh}) with Brown's V^b (broken V). The second type of broken V of Brewbaker's (V^B) is re-designated here as V^{bd}, or "broken V—dominant," in respect of the dominance it expresses over V-point (V^p).

Among the adopted symbols are: (1) V^f for the filled V phenotype which has been referred to previously as V^{wi} (V with white inside), V^p and V^{fi} (filled-in V); (2) V^{bc} for basal V (Brewbaker[2], Figure 8*E*), in place of the designation V^L to avoid confusion with Brown's V^l (full V—low), it being evident that the basal V (V^{bc}) and full V—low (V^l) types are readily distinguishable, although definitive crosses have not been made; and (3) V^{fg} is proposed here for the filled V with green base noted by Pandey.

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STALIN AND MICHURINIST AGROBIOLOGY

(*Continued from page* 56)

wheat. Yet, when, after two or three autumn plantings, this wheat begins to form seeds (the embryos of new organisms), individual grains in its ears are found to be not of the maternal species, but of a new and different botanical species. In the case under discussion an ear of hard wheat contains individual grains of soft wheat.

In 1949, on the basis of these experiments, a number of scientists and agronomists proved beyond the shadow of a doubt that in foothills and mountainous regions, in regions where the winters are severe, and especially in fields where the conditions are unfavourable to development, winter wheat may be converted into rye.

* * *

The Stalin-teaching that gradual, hidden, imperceptible quantitative changes lead to rapid and fundamental qualitative changes helped Soviet biologists to discover and prove the occurrence of qualitative transitions of plants, the actual conversion of one species into another.

Scientists and agronomists definitely found individual rye seeds in ears of wheat which to the naked eye showed no differences from ordinary ears of wheat.

These facts plainly show that under definite conditions of life imperceptible, hidden, gradual quantitative changes take place in the wheat plant, and lead to the rapid, sudden and open conversion of individual cells of the plant, in the given case—of the egg cells of the wheat plant—into a different species. Rye cells appear in place of wheat cells.

* * *

Thus, Joseph Stalin arms us with the powerful weapon of Marxist-Leninist theory and teaches us to understand the necessity for the unity of science and practice.

Thanks to the solicitude and attention of Stalin and the Bolshevik Party, Michurinist biology is developing under unprecedently favourable material and moral conditions.

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